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RELATIONSHIP OF PSYCHIATRY TO MEDICINE*

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"FOR where there is love of man, there is also love of the art. For some patients, though conscious that their condition is perilous, recover their health simply through their contentment with the goodness of the physician."[1] This oft-repeated quotation from the Hippocratic precepts shows the early recognition by the writer, whoever he may have been, of the importance of the relationship of mind and body.

Many physicians of today are quite willing to admit their lack of knowledge of psychiatric matters. I mention this not with the idea of condemning those who accept this state of affairs but rather that I may offer an explanation of its development. Most of my listeners doubtless received the same kind of psychiatric instruction in medical school that was given to me, a series of eight or ten lectures dealing with the symptoms of the psychoses, a visit to a state hospital and possibly to a school for the feeble-minded. In these institutions we saw clinical demonstrations of the most exaggerated types of the psychoses and feeble-mindedness. Most of the monstrosities in the institution were revealed for our edification. Not infrequently these demonstrations assumed the aspects of a circus side show. Such clinics did little more than develop in the minds of the students an attitude of futility in attempting anything therapeutic for such social outcasts. In some of us such performances engendered a feeling of sympathy for the patients; in others, much as they may have tried to avoid it, a sensation of disgust. Of course, teaching of the sort described is poor pedagogy. Professors of psychiatry were not wholly to blame for this type of instruction. We are well aware of the fact that deans of medical schools are constantly under pressure to allot more time to the various specialties. Psychiatry has, for many decades, been the stepsister of medicine. The psychoses are not of themselves diseases which cause death, and many of them are essentially chronic in nature. The average practitioner probably will not see many cases of the frank psychoses. But what he will encounter is a multitude of borderline cases and neuroses. Therefore with the situation as it has been, requests for the teaching of a somewhat nebulous subject were usually of little avail.

During the past five years, under the guiding hand of the National Committee for Mental Hygiene, a determined effort has been made to set up minimum standards of psychiatric instruction in the medical schools of the United States. Instead of waiting until the last year to begin instruction in psychiatry, many schools are taking cognizance of a fact which I believe to be of paramount importance, namely, that the teaching of this subject must be started in the first year with basic instruction in medical psychology, in exactly the same way anatomy and physiology are taught as preliminaries to other clinical subjects. To a certain extent it becomes a question of conditioning a student to a receptive attitude.

In a recent article, Dr. William A. White[2] has summarized the problem in his usual succinct manner, "The real difficulty is in translating psychological mechanisms into terms that are immediately comprehensible to those who have been engaged in studying the usual problems of what I call by contrast, somatic medicine."

To many physicians psychiatry consists largely of the hospitalization of mental patients for the protection of both the patient and society. So far as hospitalization is concerned, psychiatry represents the most conspicuous example of state medicine. In the United States more than 96 per cent of the hospital beds for mental patients are under governmental control. To give you a correct picture of the hospital problem in Massachusetts, I quote from the Annual Report of the Commissioner of Mental Diseases for the year ending November 30, 1934. In the twelve mental hospitals under the jurisdiction of the department and in the institution for epileptics at Monson there were 20,283 patients under treatment. In the three state schools for the feeble-minded there were an additional 4,933 patients. In all the mental institutions of the state there were 28,952 patients. On the books of these hospitals there were 31,501 patients. In the fiscal year 1934, 6,824 patients were admitted, of whom 5,304 were new admissions and 1,520 were re-admissions. With the majority of patients in a poor or marginal economic status, it is not difficult to understand why the victims

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of frank psychoses are now almost entirely a problem for state medicine. I have quoted these figures to impress upon you the importance of mental diseases as a public health problem and as a preliminary to the next step in my thesis.

Psychiatry does not deal only with hospitalized cases. One of the grave objections to the hospitalization of mental patients is the idea of relatives that the family is stigmatized by such a procedure. The utter unreasonableness of this attitude can easily be shown. It is a "throw-back" to antiquated notions that mental disease represents a punishment for sins committed. It is the old theory of diabolical possession. It also means, to some extent, a natural reaction to the more recent idea that these diseases are due to heredity. Modern studies in heredity reveal little sound or conclusive evidence in favor of this being an all-important factor in the psychoses. This is the reason for the objection on the part of careful students of eugenics to the wholesale sterilization of mental patients. Our knowledge of the major etiologic factors in mental disease is not so all-inclusive as to permit us to be too dogmatic in our statements, but certainly there is no more basis for a relative's feeling a sense of shame when a mental disease develops in the family than when any other kind of disease occurs.

One method of helping the situation is through the use of psychiatric wards in general hospitals. The Massachusetts General in Boston and the Presbyterian Hospital in New York are examples of this trend. Many city hospitals have their psychiatric departments, the outstanding examples in this country possibly being Bellevue in New York and the Philadelphia General. In these institutions splendid co-operative work between various departments has resulted in a better understanding of problems which have bothered physicians for years.

Dr. Helen Flanders Dunbar[3] in her book "Emotions and Bodily Changes" reviewed 2,251 articles and books published between 1910 and 1933 dealing with psychosomatic relationships. Even in this splendid work the literature is by no means exhaustively surveyed. Many clinicians are realizing the importance of considering the patient as something more than an aggregate of cells and are writing stimulating, thought-provoking articles on the subject. Dunbar says, "Throughout all the phases of biological research there is evidence of the same transition from the premise that the whole could be understood by a study of the parts to a new emphasis on the whole."

As a result of her investigations Dunbar noted that thinking among physicians regarding psychosomatic relationships falls into three groups: first, the idea that all psychic manifestations are brought about by an organic condition; secondly, the antithesis to this thought, overlooking the organic condition in favor of a psychic etiology; and finally, the acceptance of the presence of both psychic and organic pathology in a given patient. This is the psychosomatic point of view, with its emphasis on the concept of the organism as a whole.

As evidence of the first type of thinking, the development of general paresis is often given. We have in this disease a definite etiology, the *Treponema pallidum*. It operates in a destructive way on the frontal lobe of the brain. Many years ago Osler said that to know all there is to know about syphilis is to know all medicine. The mental symptoms of dementia paralytica are as protean as are the physical manifestations of lues. One patient may be maniacal, another depressed, one may develop a paranoid reaction type, another be expansive, one may rapidly deteriorate and another rapidly recover. Why should these patients with the same etiologic factor develop such varying clinical pictures? Many things enter into the picture and researches are being made to clarify this problem. We have, however, sufficient information to make us believe that the major reasons for the various symptomatologies present are to be found in either the original physical endowment of the patient or the effects of conditioning brought about by environmental factors.

The second type of thinking, in which the psychic factor is the only etiologic cause in producing disease, receives little following among physicians. Among quacks, charlatans, and adherents of Christian Science and other cults, however, it does assume importance. We cannot get away from the fact that many so-called "cures" follow the ministrations of these groups.

The danger of falling into the fallacious type of reasoning, "after it, therefore because of it," is in marked evidence here. Most physicians have, at some time in their careers, been guilty of this same error in reasoning. Medicine is filled with it. The healing power of nature must always be taken into consideration in evaluating therapeutic remedies. It should also be remembered that the statistics of the early hospitals accepting and practicing the Hahnemannian principle of "like cures like" were much more satisfactory than those of the allopathic practitioners, prior to their therapeutic reforms. One wonders if the personalities of the old homeopaths did not have much to do with their therapeutic triumphs and whether they did not practice an unconscious form of psychotherapy.

Among psychiatrists of today there seems to be a general acceptance of the concept of the organism as a whole. They believe that disease cannot be considered entirely in terms of either the organic or psychic, but that to understand fully the clinical picture the organism must be

considered as a unit. Both psychic and somatic factors must be evaluated. Disease is not only a pathologic process but it is an experience in life and, like all experiences, we just adjust to it emotionally. This adjustment in the case of a minor pathologic condition is relatively easy, but when we think of this emotional adjustment in a case of tuberculosis or cancer, the matter becomes more difficult and complicated. And yet this emotional adjustment affects the pathologic situation.

The pediatric and psychiatric departments of the Johns Hopkins Hospital have been working in close co-operation for several years, with very satisfactory results. The study of these emotional problems requires a considerable investment of time on the part of the physician for their solution. A co-operative study by internist and psychiatrist would divide this labor and give better therapeutic results than could be attained by either, working alone.

As a concrete problem, take the subject of enuresis. Many children suffering from this condition have no demonstrable organic lesion to account for their difficulty. The diagnosis is frequently enuresis, cause undetermined. These cases require many hours of patient investigation and hours of training before the unhappy habitual tendency of the patient is overcome. This is what is meant by looking at the problem from the concept of the organism as a whole. What of the reaction of the patient to environmental factors? What of the patient's mental response to exogenous or endogenous stimuli and the physiologic changes which we know occur in the organism as a result of these stimuli? What of the factors which occurred in the child's past which may have played a rôle in the development of the enuresis? Is the enuresis an attention-gaining mechanism? Is the child unconsciously or consciously punishing the parents? These are only a few of the questions which should be answered in studying the patient when bearing in mind the concept of the organism as a whole, the psychosomatic approach.

Schwarz[4] considers enuresis as only one factor in a complicated psychic situation; therapy of the condition becomes prophylaxis against a later neurosis of which this symptom may be the first evidence, both being produced by the same emotional disturbances. If this be true, what an opportunity our pediatricians have to do sound, preventive psychiatric work.

It is true, however, that enuresis may be cured by local measures. It does not necessarily follow that the enuresis is always due to a local condition, and by the same token it need not be due to a psychic condition. Both sides of the problem should be investigated.

That emotional disturbances may affect laboratory findings is now a matter of common knowledge. One of the tests which is commonly affected is the basal metabolic rate. It has been our experience that we are hardly ever warranted in accepting at face value a first determination of the rate of oxygen consumption. A patient may give all indications of being in a true basal state so far as external appearances are concerned and yet may be in a turmoil of seething emotions internally. Under such circumstances a patient with a markedly lowered basal metabolic rate may give an entirely misleading normal rate.

As pointed out by Ziegler and Levine,[5] working with psychoneurotic war veterans, more than ten years ago, "To the casual observer the majority of these patients would have been considered during the test as lying still on the bed. Furthermore, some patients who showed increase in metabolism and practically no objective reactions were not aware of any emotions whatsoever. The mere fact of a patient lying quietly in bed is no assurance of a state of rest." From our experience with both mentally normal and abnormal patients we can attest to the value of this implied warning.

Emotional glycosuria is a not uncommon phenomenon and the diagnosis of diabetes mellitus has been made more than once because of lack of consideration of this emotional factor. Anxiety tends to produce an increase in blood sugar. Several authors have emphasized the necessity for more adequate consideration of the psychic factors in diabetes mellitus. There seems to be little doubt that people of the so-called "nervous" temperament are more prone to develop diabetes than are others of a more phlegmatic temperament. There are many interesting cases in the literature of significant improvement in patients placed under conditions of psychic quiet and exacerbations when undergoing psychic excitement.

There are many physical conditions in which a psychic component is well recognized. It might be of some value to review some of these briefly as a means of emphasizing the importance of the concept of the organism as a whole and the therapeutic implications.

In a study of five thousand cases of Graves' disease Bram[6] found that approximately 90 per cent presented a clear history of psychic trauma which significantly preceded the disease. In 4 per cent a history of general or focal infection was the possible exciting cause of the disease. In 3 per cent the ingestion of iodine or thyroid extract was regarded by the patients as the cause of the symptoms. Bram felt that in most cases where infections appeared to serve as the exciting cause and in those cases in which no exciting cause was discovered, an undisclosed psychic trauma may have preceded the syndrome. "It appears that inherent predisposition is a prerequisite to sensitivity to psychic

traumata." He likewise states that "Individual sensitivity to psychic traumata varies qualitatively and quantitatively."

The removal of the psychic precipitating factor, combined with rest, is often sufficient to effect a cure without recourse to surgery. Crile's method of anoci-association in which he "steals" the operation is a concession to the importance of the psychic factors in operative treatment of the condition. There are authentic cases in the literature of cure of exophthalmic goitre by means of psychoanalysis. I have seen a physician friend lose many pounds in weight, develop tremors, and show marked acceleration in oxygen consumption accompanied by enlargement of the thyroid gland who, when the psychic precipitating factor was no longer operative, promptly lost all symptoms and made a complete recovery.

The heart is another organ which is particularly subject to the stresses and strains of emotional life. Attaching a subject to a cardiotachometer and giving him a simple problem of addition to solve is often sufficient to increase the heart rate twenty or thirty beats per minute. Even the anticipation of a task is often sufficient to produce a marked acceleration in the heart rate.

In cardiac patients, as emphasized by numerous authors, psyche and soma are particularly closely intertwined. A correct analysis of the patient's psyche may mean the difference between a cardiac neurotic and a continued useful citizen. We are all familiar with the disastrous results which may happen to a case of angina pectoris if exposed to a trying emotional situation. Considerable work has been done with hypnosis in trying to determine psychic factors in the precipitation and perpetuation of hypertension. Several authors have expressed the belief that an experience which is no longer in consciousness may produce the same sensations as at the time of its first happening. L. A. Conner's[7] excellent articles dealing with psychic factors in cardiac conditions contain practical advice worth acting upon.

Bronchial asthma has taxed the patience of most physicians. The number of remedies still used in its treatment make the inquiring physician wonder if psychotherapy may not serve as a useful adjuvant to drug therapy. O. Fenichel[8] believes the behavior of these patients, apart from their asthmatic symptomatology, to be that of a compulsion neurosis. Moos[9] reports the recovery of sixteen asthmatics with disappearance of Charcot-Leyden crystals and Curschmann's spirals and a return to normal of the blood eosinophil count following psychotherapy. Exposures of these patients to the supposed specific allergen did not result in further attacks.

The intimate relations of psychic factors and gastrointestinal pathologic conditions have been recognized for centuries. It has only been during the past decade, however, that psychiatrists have worked with internists in clinics in co-operative efforts to remedy the conditions. "Nervous dyspepsia" in the 1880's was a well-recognized clinical entity. It is a matter of common knowledge that anxiety or worry will cause exacerbations of gastric ulcer. In these nervous dyspepsias, so-called, we always find psychic concomitants. It is as essential in the proper treatment of these conditions to treat the accompanying psychologic condition as it is to place the patient on dietary or drug treatment. Dreyfus[10] believes that nervous dyspepsia is not a gastric disease, that the gastric symptoms, although they may completely dominate the picture, are of a secondary nature.

Cushing's[11] comparatively recent work on the relationship of peptic ulcer and the interbrain is thought-provoking, if not all-inclusive. He concluded in part on the basis of his experiments as follows: "So it may easily be that highly strung persons, who incline to the form of nervous instability classified as parasympathetic (vagotonic), through emotion or *repressed emotion*, incidental to continued worry and anxiety and heavy responsibility combined with other factors such as irregular meals and excessive use of tobacco, are particularly prone to have chronic digestive disturbances with hyperacidity often leading to ulcer, effects wholly comparable to those acutely produced."

Another gastrointestinal condition which has baffled many competent internists is mucous colitis. Most gastroenterologists are agreed that nervous manifestations are usual concomitants of the condition. Thorough investigation of these patients will usually reveal a close relationship between a trying psychologic situation and the onset of symptoms. Psychotherapy frequently is effective in the treatment of this disorder.

Both spastic and atonic constipation are frequently accompanied by neurotic manifestations. Visceroptosis has its psychic components. It is interesting that the incidence of nephropexy and fixations of various sorts are steadily declining. Surgeons are realizing that there is something more to the problem than "hitching up" a fallen viscus.

Nearly every experienced obstetrician recognizes the value of suggestion in hyperemesis gravidarum. The psychic difficulties present in most of these cases are of a rather superficial type and usually respond satisfactorily to such simple psychotherapeutic measures as suggestion. Naturally this statement does not hold true for patients who have been permitted to vomit for months and are in a highly toxic state.

A condition not infrequently encountered by

the genito-urinary specialist is impotence. By far the majority of these cases have a predominantly psychic origin. Diuresis is a frequent concomitant of anxiety states. One of the most common disturbances the gynecologist is called upon to treat is dysmenorrhea. In the last edition of Merck's Manual, some thirty-five remedies are suggested for this condition. The fact that many of these remedies are highly effective the first time used but nearly worthless the next time should make us think of the probability that the therapeutic efficacy of our remedy is based essentially on the power of suggestion. Benzyl benzoate, viburnum prunifolium, cimicifuga, ovarian extract, all have their adherents, but I have often wondered if a little water flavored with asafetida plus a liberal supply of suggestion as an adjuvant would not make a better remedy. Please do not misunderstand me in this matter; there are types of dysmenorrhea in which I am convinced that suggestion would be about as valuable in treating the condition as nothing at all.

In 1930 Derby,[12] speaking of ocular neuroses, made the observation that in his opinion ophthalmologists produce more neuroses than they cure. He says, "Too often the neurotic patient is dismissed or got rid of with a minor change in his prescription, when what he really needs is a careful analysis of his condition and an explanation of how his various aches and pains should be interpreted and treated." He believes that in 95 per cent of the patients who are constantly coming to ophthalmologists for changes in glasses, a trained psychologist is not needed for either diagnosis or treatment. He believes that the ophthalmologist can handle the neurotic condition. In his experience, fear of injuring the eyes is the major factor, and the desire to escape unpleasant situations occurs in fewer cases. Again the fact that minor changes in the prescription produce a temporary cure is a clue to the actual situation. Desire for attention is another factor which must be given consideration in this type of case. Several authors have commented on the occurrence of certain cases of conjunctivitis which resist therapeutic measures because of their essential psychic etiology. The influence of pathology of the eye on the psyche is much more important, perhaps, in the majority of cases than the influence of psychic factors on the development of eye conditions. Both, however, should be considered. Hysterical amblyopia is met with occasionally. The treatment of this condition is usually rather easy for a trained psychiatrist. It should be remembered that the mere clearing up of the symptom, however, which can be done relatively easily by means of suggestion or hypnosis, is not the whole story. It usually is necessary to probe deeper and to get at the fundamental etiology of the condition in order to effect a permanent cure. Otherwise the original symptom will often be replaced by another.

Psychogenic deafness and other psychogenic ear disturbances are relatively common, and a very large literature has developed in this field. In the field of dermatology more and more attention is being paid to psychogenic factors in the understanding and treatment of these conditions. It has been demonstrated repeatedly that skin blisters can be produced hypnotically, with the exclusion of all sources of error. All dermatologists recognize obvious psychogenic types of pruritus and urticaria. The psychogenic element entering into dermatology should perhaps receive more consideration than it has in the past. In Dunbar's book some thirty-seven pages are devoted to abstracts of the literature pertaining to psychic factors in the production of skin conditions.

It will be recognized in this discussion that I have said very little that is new. I have selected rather obvious illustrations intentionally from the various specialties of medicine. Many specialties are not even considered, although an extensive literature is available for those who are interested in nearly every field of medicine. All of us, while students in medical school, received definite instruction to "treat the patient and not the disease." It is my contention, however, that to this ideal most of us render little more than lip service. It takes time to delve into the trials and tribulations of our individual patients, and to many of us, often because of our earlier training, it seems rather futile to waste our time in listening to long-drawn-out descriptions of physical ailments. Too often do we label these patients neurotics and treat them with placebo therapy. In my opinion this is one, and perhaps not the least important, reason why many of the neurotics find themselves in the hands of various cultists and charlatans. Would it not be far better for us to practice psychotherapy in these cases intelligently than practically to invite their going to unscientifically trained people for their treatment. If we are unable either by temperament or because of the time factor to treat these patients ourselves, would it not be better to refer them to our confrères, the psychiatrists. It is my carefully considered opinion that no physician, no matter what specialty he represents, will not be able to render a better type of medical service provided he adds to his therapeutic armamentarium a basic knowledge of medical psychology.

Dr. Miller,[13] in his presidential address to the American College of Physicians, spoke on "The Changing Order in Medicine." Coming as that does from one of the most distinguished physicians in the country, his comments relating to psychiatry as it touches on medicine will bear repeating *in extenso*. May I quote him verbatim.

"Perhaps the lack of intimate personal contact which we have lost in the passing of the family physician is a factor in the situation. But whatever may be the explanation, is it not true that we, as physicians, under the influence of our passion for modern and exact science have lost sight of those more intangible and less demonstrable factors which we call the nervous and the psychic? Not only in the prevention of actual nervous or mental disorders is this factor important but also in the treatment of our patients suffering from any disease, particularly the chronic disorders. A better recognition of the importance of their mental processes would appear to be demanded which would bring us back to a fundamental appreciation of our patients as individuals rather than as cases of disease, and to an understanding of their personalities and the influences under which those personalities have developed and are manifested. Is it not at least partly true that the extraordinary development of cults of faith and mental healing in this country, estimated to affect approximately ten millions of our people directly or indirectly, may be attributed to a lack of interest in and appreciation of these factors by the practising medical profession? Personally I have a very strong feeling that as a whole we of the profession are constantly losing magnificent opportunities not only to help our patients get well but also to control and develop their characters and personalities as evidenced by their nervous and emotional as well as their intellectual responses. In other words, have we not been affected too much by materialistic and scientific progress and failed too often to appreciate the importance of the individual as a whole, which would include all of these psychic, emotional and ethical values in his life as well as the physical?"

With a continuation of the emphasis in medical schools on psychosomatic relationships, much may be hoped for in an improvement in the relationship between the physician and the patient. Perhaps no better way of emphasizing this point and concluding this discussion can be had than to repeat part of the paragraph attributed to the great "Father of Medicine" with which I opened this discourse, "For some patients, though conscious that their condition is perilous, recover their health simply through their contentment with the goodness of the physician."

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THE BLOOD IODINE LEVEL, BEFORE AND AFTER SUBTOTAL THYROIDECTOMY FOR HYPERTHYROIDISM*

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THE history dealing with the relation of iodine to the thyroid gland should be familiar to those interested in goitre problems. From the earliest records which reveal that the Chinese used a concoction made from burnt sponges on their enlarged necks, to the last decade in which iodine has become a medicinal specific in the treatment of hyperthyroidism, certain landmarks may now be viewed as fundamental in our present conception of the association of the iodine metabolism to the structure and function of the thyroid gland.

Following Courtois' discovery of iodine in seaweed, Coindet in 1820 suggested that this element was related to the function of the thyroid gland. Iodine insufficiency as a goitrogenous factor was noted by Chatin in 1849. As a result of the unfortunate reception of Chatin's work by the French Academy in 1860, the hypothesis that an association existed between iodine and goitre fell into disrepute. Interest was revived in the subject in 1895 when Baumann[1] brilliantly demonstrated the purple vapor of iodine secured from thyroid tissue. From this time, a search was instigated for specific iodine compounds in the thyroid gland. Not until 1916 was the objective attained by Kendall[2] who isolated thyroxine. In 1911 Marine and Lenhart[3] established the relation of hyperplasia of the thyroid gland to clinical hyperthyroidism and advocated the use of iodine in this condition.

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However, the fear of possible unfavorable effects due to the use of iodine was difficult to eradicate. Existing prejudice was overcome by Plummer and Boothby[4] in 1924 and the fact established that iodine medication as a prerequisite to subtotal thyroidectomy for hyperthyroidism is a safe and wise procedure.

With the above developments, the necessity for information concerning the presence and amount of iodine in the different tissues of the body became apparent. At the request of the Swiss Goitre Commission, von Fellenberg[5] in 1923 reviewed all the known methods of microchemical analysis of iodine and devised the first adequate method for estimating iodine in amounts such as are present in blood. The thoroughness of von Fellenberg's work is evident when one realizes that most existing methods of blood iodine analysis incorporate all or a part of his original procedure.

Veil and Sturm[6] and Lunde and his coworkers[7] were the first to attempt to correlate the blood iodine level with clinical hyperthyroidism. Cattell[8] reported the effect of the administration of iodine by histologic and chemical analyses of the excised thyroid tissue from hyperthyroid cases. The results of the preceding studies and of many others along related lines have demonstrated that in hyperthyroidism there is a progressive depletion of the iodine content of the thyroid gland concomitant with an increase in the iodine of the blood and urine. In our series of over 100 cases of hyperthyroidism with an elevated blood iodine, urine analysis has shown that these patients may excrete from 0.25 to 1.00 milligram of iodine per day. Since the average diet contains considerably less than 0.25 of a milligram of iodine, it seems likely that a hyperthyroid individual with an elevated blood iodine is in a state of negative iodine balance. This hypothesis has recently been more securely established by Cole and Curtis.[9] It is therefore reasonable to hypothecate, that should a hyperthyroid syndrome persist in an individual, the thyroid gland will eventually become depleted of its store of iodine at which time the blood and urinary iodine return to normal. The length of time required to bring about such a response is dependent upon the duration of the disease, together with the size of the thyroid gland at the initiation of the hyperactivity. Correlation of the results of blood iodine analysis previously reported,[10] as well as our more recent studies on the urinary excretion of iodine in cases of hyperthyroidism, lends credence to this view.

The purpose of the present study was to determine the influence of subtotal thyroidectomy on the blood iodine level and to ascertain, if possible, whether an elevated and a normal blood iodine were each a definite state of iodine metabolism in the hyperthyroid individual. Accordingly the blood iodine level was followed postoperatively in 141 cases in which the preoperative level had been determined.*

Of the 141 cases of hyperthyroidism that have been followed postoperatively, 131 cases were followed for 3 months, 81 cases for 6 months, 46 cases for 9 months, and 31 cases for 1 year. Since only 4 of the 83 cases with an elevated preoperative blood iodine did not show a *decrease* in blood iodine postoperatively and since only 4 of the 58 cases with a normal preoperative blood iodine did not show an *increase* in blood iodine postoperatively, it was considered permissible to group the cases, dependent upon the preoperative blood iodine level. Furthermore, it seemed fair to average the blood iodine values of the 2 groups, since only the 8 cases cited, of the 141 cases followed, did not show a characteristic blood iodine trend. The results are shown in the following table:

GROUP I. Cases of hyperthyroidism with an *elevated* preoperative blood iodine:

		Number of Cases Followed Postoperatively to:			
	Preop.	3 mos.	6 mos.	9 mos.	1 year
No. of cases	83	76	39	21	14
Average blood iodine in micrograms per cent	26.0	7.7	7.0	7.6	7.0

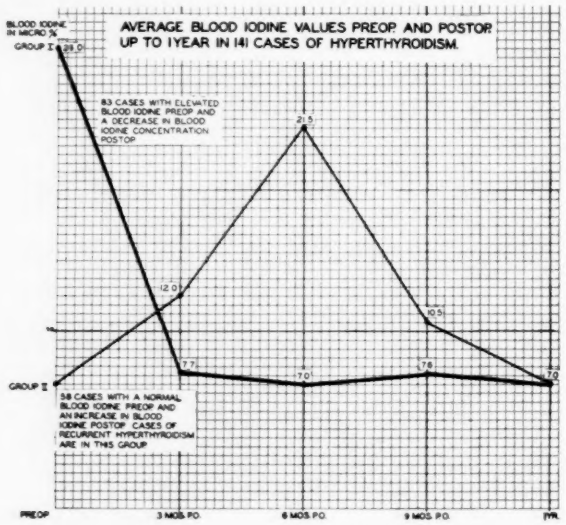
GROUP II. Cases of hyperthyroidism with a *normal* preoperative blood iodine:

		Number of Cases Followed Postoperatively to:			
	Preop.	3 mos.	6 mos.	9 mos.	1 year
No. of cases	58	55	42	25	17
Average blood iodine in micrograms per cent	7.0	12.0	21.5	10.5	7.0

From the table it will be seen that the average blood iodine level of hyperthyroid individuals with an elevated preoperative blood iodine (Group I) returned to normal 3 months postoperatively and throughout the time followed was found to remain normal. In the hyperthyroid cases with a normal preoperative blood iodine (Group II), the average blood iodine value was increased 3 months postoperatively

*The blood sample for iodine analysis[11] was taken the morning following hospitalization. Patients who had received iodine within a two-week interval prior to admission were excluded from the present report. Preoperative Lugol's was given for 8 to 15 days and for 1 week postoperatively. When stage operative procedures were found necessary, the patients received iodine daily till the completion of the last stage, the interval between operations being 6 weeks. No iodine was given after final discharge from the hospital. At 3, 6, 9 and 12 months postoperatively, the patients were hospitalized overnight, the clinical condition checked and a blood sample taken for iodine analysis.

and reached a peak of elevation at 6 months, decreasing at 9 months, and returning to normal at 1 year. The cases followed for 1 year are characteristic of the above blood iodine trends, so it is believed that the cases not followed for the entire time period will eventually show the same blood iodine response. The characteristic trend of the blood iodine level in the two groups is shown graphically in the accompanying chart.


AVERAGE BLOOD IODINE VALUES PREOP AND POSTOP UP TO 1 YEAR IN 141 CASES OF HYPERTHYROIDISM.
BLOOD IODINE IN MICRO %
GROUP I
83 CASES WITH ELEVATED BLOOD IODINE PREOP AND A DECREASE IN BLOOD IODINE CONCENTRATION POSTOP
21.5
12.0
7.7
7.0
7.6
7.9
10.5
GROUP II
58 CASES WITH A NORMAL BLOOD IODINE PREOP AND AN INCREASE IN BLOOD IODINE POSTOP. CASES OF RECURRENT HYPERTHYROIDISM ARE IN THIS GROUP
PREOP
3 MOS. P.O.
6 MOS. P.O.
9 MOS. P.O.
1 YR.

The apparently significant clinical findings in relation to these results were as follows:

In the hyperthyroid cases who had an elevated blood iodine preoperatively (Group I) and a postoperative fall in blood iodine, there was no evidence of recurrent or persistent hyperthyroidism in any case within the time followed.

In the hyperthyroid cases with a normal blood iodine preoperatively (Group II) and a postoperative rise in blood iodine, 4 cases have been diagnosed clinically as having recurrent or persistent hyperthyroidism and 3 cases as borderline recurrence to date—2 cases have increased exophthalmos since operation but the thyroid is apparently negative.

If the blood iodine be considered as an index of the amount of thyroxine in the blood, the results suggest that the iodine hormone, thyroxine, is not the sole factor in hyperthyroidism—a hypothesis based on 2 cases of recurrent hyperthyroidism that were followed untreated and showed at the 9 and 12 month postoperative examination increased signs of recurrent hyperthyroidism in association with a fall in blood iodine to the original normal preoperative level.

The present results suggest that subtotal thyroidectomy in the early stages of hyperthyroidism is inhibitive of recurrence of the syndrome. Or, when the blood iodine is normal, a more radical subtotal thyroidectomy may reduce the incidence of recurrent hyperthyroidism. However, the possibility should be considered that, since the thyroid tissue removed from the latter type of case generally showed a characteristic histopathology[12] (irregular involution and Hürthle cells),[13] regeneration of such tissue left at operation may give rise to an increase in the blood iodine level together with the manifestations of recurrent hyperthyroidism.

In conclusion it may be stated that, on the basis of these results, the blood iodine level is indicative of a state of iodine metabolism and, when influenced by abnormal thyroid function as in hyperthyroidism, is of interpretable value, both before and after subtotal thyroidectomy.

SUMMARY

1. Pre- and postoperative blood iodine studies are reported on 141 cases of clinical hyperthyroidism.

2. Hyperthyroid cases with an elevated preoperative blood iodine show a decrease to normal level following subtotal thyroidectomy.

3. Hyperthyroid cases with a normal preoperative blood iodine show an increase in blood iodine following subtotal thyroidectomy, the greatest elevation being at the 6 months postoperative period.

4. Four cases of recurrent hyperthyroidism and 3 cases of borderline recurrence were found to be in the group with a normal preoperative and an elevated postoperative blood iodine.

5. A more radical subtotal thyroidectomy in hyperthyroid cases with a normal blood iodine is suggested as a means of reducing the incidence of recurrent hyperthyroidism.

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NEW HAMPSHIRE MEDICAL SOCIETY

ACUTE GASTROINTESTINAL DISEASE IN INFANTS*

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AS one considers the advances in the saving of life and the improvement of health in this country since the beginning of the twentieth century, the reduction of infant mortality must stand among the foremost achievements. In 1915 the general infant mortality rate in the United States registration area was 100 per 1,000 live births. In 1932 it was 58. In 1900 the infant mortality rate for Massachusetts was 157 and in 1934 it was 49. If one analyzes this general rate into the specific causes of death, it is found that the reduction has occurred primarily in deaths due to diseases of the gastrointestinal tract. In 1915 the infant mortality rate in the United States birth registration area for these diseases was 20, or 20 per cent of the total infant deaths. In 1932 it was 5, or 0.88 per cent of the total deaths. In Massachusetts the rate fell per 1000 live births from 45 in 1900 to 2.7 in 1934. In 1900, deaths from these diseases were very unevenly distributed throughout the year. There was a high peak of deaths during the summer months, especially among artificially fed babies. Those of you who were in practice at that time can well remember the large number of babies who had so-called "summer diarrhea" and the high incidence of fatality. The picture has entirely changed today. The present deaths are distributed fairly evenly throughout the year. The high peak of summer mortality has disappeared. "Summer diarrhea" is no longer prevalent.

It is not possible to explain exactly or to evaluate accurately the relative importance of the factors which are responsible for the change which has occurred. It is clear, however, that three forces have exerted a significant influence upon the situation, a better knowledge of how to feed babies artificially, improvement in the care of babies, and the pasteurization of milk.

Better artificial feeding. Since the beginning of the century there has been a substantial increase in the body of knowledge concerned with the nutrition of infants. This knowledge has been acquired through painstaking laboratory investigations conducted on animals, and careful clinical and metabolic studies of the actual food intake of normal babies making satisfactory progress in growth and development. We do not know all the facts about infant nutrition and there is need for much further investigation, but our present knowledge is sufficient for reasonably satisfactory results if one applies intelligently the information which we possess. Our greatest need at the present time is to bring practice up to the present state of knowledge.

We know that there are certain food elements which are essential for the growth and development of infants. We have some conception of the quantitative optimum values of these elements and of the ranges within which variations may occur and not exceed the infant's power of adaptation. We have also learned the importance of vitamins and the significant rôle which they play in infant metabolism and in the prevention of disease. For babies who cannot receive woman's milk, the food requirements except for total calories and vitamins can be met by cow's milk given in amounts determined in relation to the age, weight, and other individual characteristics of the infant. After six months of age additional iron in some form will also be needed. It has been demonstrated clearly that the vast majority of normal infants will progress satisfactorily on simple whole milk dilutions with the addition of sugar and the necessary vitamins. Evaporated milk may be substituted when a proper liquid milk is not easily available. The kind of sugar which is used is not of great importance and the choice depends more upon the preference and experience of the physician than upon the superiority of one variety over another. Vitamin C found in orange juice, tomato juice, and other fresh fruits must be given to supplement the limited amount contained in milk. Vitamin D must also be supplied by cod liver oil or some equally potent substance to insure against the development of rickets.

The market is flooded with many ready-made patented and proprietary baby foods. The use of these foods has become widespread, especially in the last ten years. I am inclined to believe that this practice has retarded and not aided the intelligent artificial feeding of infants. In order to provide an infant with the essential food elements in adequate amounts and in proper relation one to the other, it is necessary to understand the fundamental principles of infant nutrition. The principles may be easily understood with a moderate amount of study. Once these are mastered, the adaptation of the principles to the needs of the individual baby is not a difficult problem. The use of proprietary foods is a rule of thumb procedure and presupposes no knowledge of the fundamentals upon the part of the practitioner. Lack of success with one food is often met by trial of another and sometimes of another and another. It is all too common to find a baby who has

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been changed back and forth without at any time having received an adequate food. We shall all of us have much better success in infant feeding, and the babies under our care will make better progress, if we learn the fundamental principles and acquire experience in the use of simple whole milk dilutions.

Better care. Not only do we know more than formerly about the nutritive requirements of infants, but babies receive better care today than they did thirty-five years ago. In this respect practice has approached more nearly and more widely to the application of knowledge. This has been brought about through the education of mothers and others who care for babies. Infant welfare stations, public health nurses, and many lay organizations have contributed to the educational program. As physicians we are responsible for the content of the instruction which is given by lay persons, and for the actual instruction of our own patients. We are fortunate that so many agencies in the community are fostering this work. We must continue to furnish leadership, the maintenance of high standards, and the introduction of new knowledge as it may become available.

Pasteurized milk. It is interesting to compare the falling infant mortality rate, especially in gastrointestinal diseases, with the extension in the pasteurization of milk. The two lines run very nearly parallel. The pasteurization of milk is not only effective against specific diseases, such as tuberculosis, scarlet fever, and septic sore throat, but has undoubtedly had a direct influence in the reduction of the gastrointestinal diseases of infancy. There is no evidence to indicate that the pasteurization of milk causes any decrease in its nutritive value. As physicians we must be insistent in our attitude that no baby shall be fed raw milk.

If we turn now from a consideration of mortality to a discussion of morbidity, we shall find that despite the reduction in the number of deaths, the gastrointestinal diseases or symptoms referable to the gastrointestinal tract still occupy an important place in medical practice among infants. In infants, disease is recognized by the appearance of some objective sign. In an analysis made by J. A. Johnson of 3000 consecutive admissions to the Infants' Hospital in Boston, it was found that 75 per cent of the babies were brought to the hospital because of one of three reasons, loss of weight or failure to gain, vomiting, or diarrhea. When an infant presents any one or any combination of these complaints, how should one proceed in order to determine the cause of the symptoms and to institute proper treatment?

First of all, one should secure an accurate history. It is to be remembered that in securing a history from an infant one is dependent entirely upon information obtained through a third person. The infant can give no account of his own symptoms. One must rely upon the objective signs which he presents, such as crying or irritability, or some other departure from normal behavior. The mother or the nurse may be an accurate observer, but the chances are that the unprompted history will be tinged with the point of view of the person who gives it. The fact that the mother is worried and anxious about her sick child tends to make her distort the facts and to overemphasize some unimportant details at the expense of essential data. It takes time, patience, and some experience to arrive at an orderly statement, but it is time well spent and no actual detail in the history is too trivial to bear consideration.

It is equally important after securing as reliable a history as possible, to make a careful and complete physical examination. Here again, as in history taking, the technic is somewhat different from the procedure in dealing with adults. It is very difficult to make a satisfactory examination of an infant who is frightened and rebellious. Therefore, a certain amount of time must be spent in gaining the confidence of the baby, and it is important to conduct as much of the examination as is possible before the baby is disturbed. It is remarkable how much information may be acquired from simple inspection. The next stage of the examination should be undertaken by doing first the things which are least likely to cause an unfavorable reaction. Babies always resent quick, sudden movements. They object to cold hands. It is desirable to use a stethoscope early before crying begins, and to postpone, until the last, looking at the throat and a rectal examination.

With an accurate history, a careful physical examination, and whatever additional laboratory data are necessary, one is in a position to consider a diagnosis. Again referring to the experience at the Infants' Hospital one may divide into four groups the conditions which are most likely to cause the presenting symptoms of either vomiting, diarrhea, or loss in weight, or any combination of these. There will be a few infants who present other conditions, such as intussusception, but in the great majority the diagnosis will be found to be in one of these groups:

1. Infection.
2. Congenital anomalies.
3. Faulty environmental or hygienic conditions.
4. Improper food.

Any one infant may fall into more than one of these groups at the same time, but the relative frequency and importance of the causes of the symptoms of vomiting and diarrhea or loss of weight are in the order given.

Infection. Infection in infants may give rise to gastrointestinal symptoms not only when the infection is in the gastrointestinal tract, enteral infection, but may also when the infection is in some part of the body, parenteral infection. In any child who has an acute gastrointestinal disturbance one must search thoroughly and repeatedly for evidences of infection. It is important to point out that infection in infants frequently is manifest by quite different physical signs from those in adults. The temperature may be normal or even subnormal in the presence of severe infection. The white blood count, however, is usually elevated but may be normal with a relative increase in the polymorphonuclear leucocytes. When there is disease of the respiratory tract, which is one of the most frequent types of infection, the only signs evident on physical examination may be an abnormal reddening of the ear drums. The physical signs of pneumonia early in the disease may consist only of a suppression of breath sounds over the affected lobe, or may show no abnormal physical signs in the chest. The roentgen-ray is of great assistance in diagnosis at this stage of pulmonary involvement. The reflexes in infants are relatively unreliable and even in meningitis there may be no rigidity of the neck and an absent Kernig's sign.

The definite signs of gastrointestinal tract infection, mucus, pus and blood in the stools, may not make their appearance until the disease has been present for several days. In the milder types of dysentery there may be no bleeding. Positive agglutination reactions in the blood will, however, indicate the specific character of the infection. One is not so likely to miss this form of infection as to attribute to disease in the gastrointestinal tract an infection elsewhere in the body with gastrointestinal symptoms.

Congenital anomalies. A congenital anomaly is missed in diagnosis, in most instances, not because a diagnosis cannot be made but because of failure to consider this possibility and to look for it. As in infection, because the presenting symptoms are of the gastrointestinal tract, it does not necessarily follow that the congenital anomaly which causes the symptoms is in the gastrointestinal tract. For instance, an anomaly of the urinary tract which is not at all rare in infants may give no signs of urinary symptoms but may be the basis of persistent vomiting and diarrhea. Particularly is this true if there is a superimposed or concurrent infection. Examination of the urine of a sick infant should be considered a routine part of the physical examination. Congenital heart disease may explain failure to gain, or loss in weight, even though there are no signs of cardiac decompensation. Congenital lesions of the gastrointestinal tract itself are not rare and should always be considered in the diagnosis of any sick infant, especially when vomiting is an important symptom. The earlier in life symptoms of disease appear, the greater the chance that some congenital anomaly is present, but the late appearance of symptoms by no means excludes an anomaly. An intermittent occurrence of symptoms is also consistent with a mechanical explanation on the basis of an anomaly in development. The methods by which an anomaly may be proved to exist vary with the type of lesion, but in every instance require careful examination and often the use of supplementary special and laboratory procedures, especially the roentgen ray.

Faulty environmental or hygienic conditions. High temperature, especially when associated with high humidity, is universally recognized as having an unfavorable effect upon infants, but, further than that, this group of causes is often given little consideration. Unlike infection and congenital anomalies, for which a careful physical examination is of such great importance, in these conditions the facts are obtained only through a careful history. History taking is time-consuming and, in order to secure the necessary information, must be meticulous in detail. We must know how the food is prepared, how it is given, the hours of sleep and daily routine, and many other fine points of the infant's care. Many babies are sick because of poor care and many can be cured by the institution of proper hygiene. It is a common occurrence for all symptoms of disease to disappear when an infant is admitted to a hospital and placed upon a good routine. A good infant's nurse may often accomplish the same results even though the infant remains at home.

Improper food. When an infant has a disturbance of the gastrointestinal tract one might suppose that the most probable explanation would be found in improper food, yet we know from experience that this is the least likely cause for the symptoms. We are driven to conclude that an infant has a wide tolerance in digestive and assimilative capacity and can adapt himself to a variety of food combinations provided only if his fundamental requirements are satisfied. From a practical point of view for us as physicians, it is important to remember that other causes for digestive symptoms in infants are to be sought beyond the food intake. It is not enough to change the formula or to try some other or new proprietary baby food. We will rarely cure vomiting or diarrhea that way. One should, of course, know what food a baby has been taking and if it is grossly faulty the proper food should be substituted, but the cause of the acute illness, even though the symptoms are primarily or entirely confined to the gastrointestinal tract, will usually be found to be other than improper food. This is not to be interpreted to mean that faulty feeding is never a

cause of disease in infants, because of course such an impression is contrary to the facts. Lack of vitamin C in the diet may cause scurvy, or a deficiency of iron may result in serious anemia, but improper food is less often the cause of disease than is commonly believed, and of less importance in explaining gastrointestinal symptoms in infants than either infection, congenital anomalies, or improper environment and hygiene.

Diagnosis. When an infant is taken sick with vomiting or diarrhea or both, or fails to make satisfactory progress in weight, we may reasonably expect to find one of four things, or some combination of these four things, to be the cause. In the order of frequency these things are the following:

1. Infection somewhere in the body, not necessarily or most likely in the gastrointestinal tract.
2. A congenital anomaly again not necessarily in the gastrointestinal tract.
3. Faulty environment or hygiene.
4. Improper food.

A careful history and a complete physical examination are necessary to determine which of these four things is responsible for the symptoms in the particular baby. If one takes enough time for the history and is sufficiently thorough in the physical examination, a diagnosis can be made in most instances.

Treatment. The treatment will vary according to the diagnosis. When infection is present the treatment may be specific or nonspecific according to the findings. Of the nonspecific measures the maintenance of body fluids at, or their restoration to, normal levels, if there has been severe loss, constitutes one of the major procedures essential to the saving of life. Studies indicating the specific change in blood chemistry brought about by vomiting and diarrhea have furnished the basis for effective therapy. Real advance has been made during the last few years in this matter. We have come to realize, also, that starvation for long periods of time is a deterrent, not a help, to recovery.

The treatment for a congenital anomaly is usually surgical. Some anomalies have thus far not been successfully dealt with, but the number is constantly growing less. The technics of the operative procedures require special familiarity with this field and skill in the handling of small and delicate tissues.

The correction of faulty environmental and hygienic conditions requires education of the family. Frequently the environment cannot be altered but much can be done to mitigate its deleterious influence. Poor hygiene can be changed but it is often a long and tedious process and requires patience and consideration.

For the treatment of an infant who has been receiving improper food, one needs to know the food requirements of the particular baby and how to provide a formula which will meet them. As has been said earlier, whole milk dilutions with added sugar and vitamins will supply everything which is needed in practically every instance. One has only to learn how to use these simple tools to be able to prescribe the proper food for a normal baby. It is rarely, if ever, necessary to employ ready-made foods, the composition of which is imperfectly understood.

In conclusion, I should like to emphasize again the importance of an accurate history and the necessity for a complete careful physical examination as the basis for an accurate diagnosis. Vomiting, diarrhea, and loss of weight alone, or in combination, are the commonest symptoms of disease in infants. In the order of importance, infection, congenital anomalies, faulty hygiene, and improper food are the causes of these symptoms. Treatment to be effective must be directed toward a removal of the cause.

DISCUSSION

CHAIRMAN KITTREDGE: Discussion of this paper will be opened by Dr. Ursula G. Sanders of Concord, New Hampshire.

DR. URSULA G. SANDERS: Dr. Smith has stressed the progress that has been made in lowering the number of cases of diseases of the gastrointestinal tract in infancy. Here, at the present time, there is surprisingly little "summer diarrhea". For the city of Concord, mortality figures for diarrhea and enteritis in children under two years give the following comparison: 1891, 28 deaths; 1910, 13; 1921, 4; 1931, 1. Statistics for the entire state of New Hampshire show a recent reduction in deaths from this cause: 1932, 32; 1933, 18; 1934, 16.

These results seem to have been accomplished largely by Public Health measures, the improvement of milk having been a major factor. In New Hampshire, milk inspection began in 1903, delegated to the town health officials. This greatly increased in scope until, in 1931, State regulations were adopted governing the process from production to distribution. General education in the better care of infants has been an important factor. In this respect, with the new programs for child welfare, one anticipates steady progress.

The importance of differentiating the causes of gastrointestinal disturbances has been emphasized by Dr. Smith. On a pediatrics service, one is familiar with the infant who has been put through a variety of formulas, when a cause for gastrointestinal symptoms other than faulty intake is later found responsible.

I wonder if allergic phenomena might not be included in listing the various conditions giving rise to gastrointestinal symptoms. In allergic reactions, the changes in the capillary mechanism may result in congestion, exudation, and hemorrhage, producing symptoms such as nausea, vomiting, abdominal colic, and diarrhea. An interesting case which might be classified in this allergic group was seen recently. Ten days following vaccination for smallpox in a child marked the onset of allergic manifestations (Henoch-Schönlein) with severe, typical gastrointestinal disturbance.

SURGICAL DISEASES OF THE ALIMENTARY TRACT IN INFANTS*

BY WILLIAM E. LADD, M.D.†

IT may not be out of place in a meeting of this sort, which is concentrating on pediatric problems, to refer to the development of pediatries as a specialty. You will recall that the problem of infant feeding was one of the principal factors in bringing pediatrics into prominence as a specialty, and like many other movements in medicine and surgery, its importance was given undue emphasis in the beginning. This statement is made with no wish to belittle the advances which have been made in infant feeding, but rather to call your attention to the fact that all disorders of the alimentary tract are not due to improper feeding, and by that same token to assure you that they cannot all be remedied by changing the character of the formula. It cannot be too often reiterated that the first step in the successful treatment of any given case is the making of a correct diagnosis. To accomplish this in infancy is often more difficult than in adolescence or adult life. The history-taking is obviously more difficult, as it must be taken from a third person, and the physical examination usually is hampered by fussing or lack of co-operation of the patient. X-ray examination and other laboratory findings should be used to supplement but not supplant the history and physical examination. The physical examination of an infant should be complete, but the order of procedure is more important than in the adult. Any part of the examination which is painful must be postponed until the end. Many an abdominal examination is made valueless by first gagging an infant with a throat stick. Everything that it is possible to learn by inspection should be first noted. In making the abdominal examination, distention, engorgement of the abdominal veins, visible peristalsis, or tumor should be looked for before the infant is touched. Gentle palpation must precede deep palpation. Percussion and auscultation should be included, and finally to be complete, a rectal examination should be made. Pathologic conditions of infancy which attract one's attention to the alimentary tract by vomiting or other manifestation, and which require surgical aid, are really more common than those which require regulation of feeding. It is my purpose to discuss some of these surgical diseases.

Congenital hypertrophic pyloric stenosis is one of the commonest. In our series of 620 cases, this condition was found to occur more commonly in the male in the proportion of 85 per cent male to 15 per cent female infants. The symptoms usually start in the third week of life, though in 10 per cent of the patients they started in the first week. Vomiting, projectile in character, and with the vomitus containing ingested food and gastric secretions only, is the first and most prominent symptom. It is important to remember that if the vomitus contains bile one is not dealing with pyloric stenosis. The stools become scanty. The infant loses weight and becomes dehydrated. On physical examination, peristaltic waves may be seen in the epigastrium running from left to right. On palpation, an olive shaped and sized tumor may be felt in the right upper quadrant near the mid-line just under the liver, or sometimes as low as the umbilicus. Success in palpating the tumor is increased by experience and by obtaining relaxation of the abdominal muscles. The simplest method of obtaining the desired relaxation is by feeding the infant and making the examination during the feeding or just after vomiting has occurred. If one finds these five cardinal signs and symptoms, namely, projectile vomiting, scanty stools, loss of weight and dehydration, visible gastric peristalsis, and the characteristic palpable tumor, the diagnosis of pyloric stenosis is almost certain and the supplementary use of an x-ray examination is neither necessary nor desirable.

The treatment is surgical, and includes careful preoperative care and postoperative feeding. Failure to include these two important parts of the surgical treatment is the cause of the still high mortality in the large general hospitals, or in the hands of surgeons unfamiliar with infants. The preoperative care consists in overcoming dehydration and ketosis and maintaining normal body temperature. The operation of choice is a pyloromyotomy. It is a simple operation, but its simplicity apparently is misleading and is the cause of too frequent errors of technic. The commonest errors are, first, failure to cut all the circular muscle fibres of the pyloric sphincter in which case the symptoms persist; secondly, perforating the mucous membrane at the duodenal end of the pylorus, which if unrecognized leads to peritonitis and a fatal termination; thirdly, failure to close the abdominal wound carefully in layers with silk

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sutures. Fatalities following evisceration in these emaciated infants have been too frequent and may be largely avoided by a suitable closure of the abdominal wound. The postoperative care of these infants is important. Administration of parenteral fluids is continued if the state of dehydration calls for it. Feeding is begun almost as soon as the patient is out of the anesthetic. Whey is given in small amounts for the first twenty-four hours, then whey and breast milk, and by the end of the second day straight breast milk in small amounts which is gradually increased up to the caloric requirement of the infant. These patients do better in their postoperative period with small feedings at more frequent intervals. It is unwise to try to get them onto a four-hour interval between feedings for at least two weeks after operation.

The surgical treatment of pyloric stenosis is among the most satisfactory procedures in the surgical field. We have cared for 620 cases at the Children's Hospital in the last twenty years. In the first 125 cases, 13 deaths occurred, a mortality of approximately 10 per cent, while in the next 495 cases the mortality dropped to 4 per cent, and in this latter group 160 patients were operated on consecutively without a fatality. The results in private practice have been a little better than those obtained in the charity wards, for the reason that as a rule such patients seek surgical relief earlier while they are in better general condition.

Before dropping the question of pyloric stenosis, a word should be said about pylorospasm. This condition is unquestionably a real one and sometimes difficult to differentiate from pyloric stenosis. The general hypertonicity of the infant, the irregularity of the symptoms and the failure to demonstrate a tumor are the most important points of differentiation.

When vomiting takes place in the first few days of life and the vomitus contains bile, one should think at once of some form of arrested development of the alimentary tract, causing obstruction below the ampulla of Vater. These conditions are far more common than is usually supposed. We have divided these cases of congenital obstruction into two main groups, the group of intrinsic obstruction and that of extrinsic obstruction. Many embryologic events take place between the fifth and tenth weeks of fetal life. At about the fifth week of fetal life the intestinal lumen becomes obliterated by epithelial concrescence to become re-established again by the tenth or twelfth week. The cases of intestinal obstruction which have been classified as intrinsic are those which are dependent on arrest in development of the intestine during its progression from the solid to the tubular stage. This arrest in development results in the formation of a blind end, causing complete obstruction, or in a velum remaining across the lumen with a small opening in it causing a partial obstruction. Such congenital atresias or stenoses may take place in any part of the intestine or may be multiple. In a series of fifty-three cases at the Children's Hospital, not including the obstruction of the sigmoid or rectum, the obstruction occurred nine times in the duodenum, seven times in the jejunum, and 31 times in the ileum, and in six cases the obstruction was either multiple or involved the colon. The diagnosis is made by the usual symptoms and signs of intestinal obstruction, namely, pain, vomiting, lack of stools, abdominal distention, visible peristalsis or intestinal patterning, shock, and dehydration. Exceptions to the rules are found in the atresias of the duodenum in which the vomiting may be sufficiently effective so that there is no distention. Laboratory data which may be helpful in confirming the diagnosis, are failure to find keratinized epithelial cells in the rectal discharge, and an x-ray of the abdomen without the ingestion of contrast media. This latter procedure will often demonstrate very accurately the site of the lesion by the position of the air in the intestine above the obstruction and the lack of it below.

The mortality in these conditions is extremely high, but the percentage of recoveries is increasing. In this series of fifty-three patients, ten have survived. Five of the nine patients in whom the obstruction was in the duodenum have recovered, two of the seven patients in whom it was in the jejunum, and three of the thirty-one who had obstruction in the ileum have recovered. Of the ten recoveries, nine resulted from a primary anastomosis by a special technic, and only one as a result of an enterostomy with a secondary anastomosis. This is important to remember as it is usually the surgeon's tendency, when confronted with a case of atresia and the collapsed minute bowel below, to think that an enterostomy instead of a primary anastomosis is the operation of choice. In reality the exact opposite has been proved to be the correct procedure. It is also desirable to know that in the cases of atresia the bowel is likely to perforate a few inches behind the blind end by the end of the fifth or sixth day. The chance of recovery is much greater if the operation is performed prior to this time, preferably by the third or at the latest, the fourth day.

The cases classified here as extrinsic obstructions are those dependent on a faulty rotation of the midgut. They all have in common the lack of the normal oblique attachment of the mesentery of the small intestine and the normal attachment of the mesentery of the ascending colon. There is quite a variety in the abnormalities found and they are apt to be extremely

confusing unless the normal embryologic development is borne in mind. As the postarterial segment of the midgut returns from the base of the umbilical cord to the abdominal cavity, going first to the left side and rotating to the right in front of the duodenum, the cecum may become attached to the posterior abdominal wall so as to constrict the duodenum where it crosses it. This of course gives the signs and symptoms of high intestinal obstruction. The whole midgut may remain unattached to the posterior abdominal wall except at the point of origin of the superior mesenteric artery. In this instance, a volvulus of the midgut is likely to take place, giving the symptoms of acute intestinal obstruction. The volvulus usually twists in a clockwise direction, though occasionally it goes in the reverse direction. In either event the pressure comes on the third part of the duodenum first, so that in the early stages of the obstruction there is not very marked general distention. The diagnosis of these conditions is made by the usual signs and symptoms of intestinal obstruction. An x-ray examination without the administration of barium may differentiate the extrinsic and intrinsic intestinal obstruction, or if the symptoms have not come on immediately after birth, a barium enema and x-ray examination may make the diagnosis definite.

The treatment of these conditions by the principles which have been devised and adopted at the Boston Children's Hospital has a high percentage of success. Prior to the adoption of the present type of operation these cases were 100 per cent fatal, in our clinic at least, and in reviewing 349 articles in the literature fairly recently, only fifteen operative recoveries were noted. In the cases in which the obstruction was due to the cecum, colon, or superior mesenteric artery impinging on the duodenum, it was found that this could be relieved by transferring the cecum with its mesentery to the left upper quadrant and exposing the duodenum anteriorly throughout its whole length. This is accomplished by making an incision in the posterior parietal peritoneum just to the right of the duodenum and pushing the cecum and colon with its blood supply to the left; in other words, restoring an earlier state of embryologic development. In the cases of midgut volvulus, the whole midgut (i.e., the intestine from the duodenum to the middle of the transverse colon) must be delivered out of the abdominal incision to find out in which direction the volvulus has taken place. The volvulus is then reduced and the procedure described above carried out. It has been found inadequate to reduce the volvulus only. This has been invariably followed by recurrence or a fatal termination or both. There have been twenty-nine of these cases of intestinal obstruction due to mal- or nonrotation of the bowel. In twenty-one of them, the type of operation I have described has been done. Of these twenty-one patients sixteen have recovered. In the other eight patients, various other types of operations have been performed with a fatal result in every instance.

As the infant gets beyond the first few days or weeks of life he becomes subject to pathologic conditions not the result of congenital malformation. Intussusception is certainly one of the most important of these to bear in mind, as the mortality is so greatly affected by the time at which the diagnosis is made. There should be no mortality from this condition if the diagnosis is made early enough and a capable surgeon is at hand. The peak of incidence is in the seventh month of life. The disease occurs most commonly in well nourished, healthy looking babies, which is presumably one reason for the diagnosis being so frequently missed at the physician's first visit when it is so important that it should be made. The onset is sudden, characterized by crying, evidence of abdominal pain, pallor, nausea, and vomiting. The pain is severe only when peristalsis is active, and between times the healthy appearing infant may be entirely contented, which is an added reason for missing the diagnosis. A normal stool may be passed in the first hours of the disease but by the end of twelve hours, blood usually appears in the rectum. The right side of the abdomen is the most common place to find the mass in the early stages of the disease, as the ileocecal region is the most common situation for the invagination to originate. A short time later the intussusception may have advanced to the hepatic flexure where it is difficult to palpate. As it advances again to the transverse or descending colon it is easily recognized until general intestinal distention becomes so marked as to obscure it. At this stage it may be readily felt by rectal examination, the advancing point feeling much like the adult cervix uteri.

The treatment is surgical. From time to time articles appear in the literature readvocating the treatment of injection of fluids into the colon to cause the reduction of the intussusception. Some of these papers show good results, but it should be noted that the good results are obtained only in that group of cases in which we have no mortality by operative treatment, namely, the cases which come to operation in the first twenty-four hours of the disease. Seventy-nine such patients have been operated on since 1928 without mortality. The injection method is uncertain even when performed under the fluoroscope. If unsuccessful, the injection necessarily adds to the shock, wastes time and increases the mortality in the patients in whom the margin of safety is small. In this series of

431 cases, thirty-two patients were operated on in the period from 1908 to 1912, eighteen of whom died, showing a mortality of 59 per cent. From 1912 to 1928 the statistics have consistently improved and in the period from 1928 to 1936, 149 patients were operated on, of whom twenty-one died, a mortality of 14 per cent. This drop in mortality from 59 per cent to 14 per cent is undoubtedly due in large measure to receiving the patients earlier, but partly due to better preoperative, operative, and postoperative care. When once the diagnosis has been made, the operation is performed without delay, but during the time the operating room is being prepared, parenteral fluids are given. In the operation as much of the reduction as possible is done intra-abdominally. In the last part of the reduction which is always the most difficult, much more patience is used than formerly, even to the extent of splitting the muscular coats of the receiving ring, in order to complete the reduction before resorting to resection. This means that fewer resections are done now than formerly. If the intestine is gangrenous, of course resection must be performed. There have been thirty-three resections done with five recoveries. When resection is necessary it is best performed in two stages, the resection and double-barreled enterostomy as the first stage, and the closure as the second stage. A high temperature and other evidences of severe toxemia usually follow the reduction of intussusception. The administration of glucose intravenously and saline by clysis appear to be the most successful means at our disposal for combating this toxemia. In this group of 431 cases there have been eight recurrences. One of the eight was a patient who had been operated on at another hospital and in whom the ileum had been stitched to the colon in hope of preventing recurrence. We do not advocate doing any of the operations that have been recommended for preventing recurrences for three reasons. First, their efficacy is questionable; secondly, they add to the danger of the operation; and thirdly, we have so far had no mortality in this group of recurrent cases. Fatalities in intussusception are preventable by early diagnosis and appropriate treatment.

This paper of course covers but a small fragment of the surgical abdominal conditions which occur in the first year of life. It is obvious that they could not all be included. These few were selected as being among the more important and demonstrating some of the principles of surgery in infancy.

DISCUSSION

PRESIDENT ABBOTT: Discussion of Dr. Ladd's paper will be opened by Dr. MacLean J. Gill of Concord.

DR. MACLEAN J. GILL: I just want to say that from the pediatrician's standpoint, I certainly agree that pyloric stenosis is a surgical problem and should never be treated medically, once the diagnosis has been made. The diagnosis is made when you have palpated the tumor. That is going to take a lot of time and a lot of patience on the part of the examiner. It may take more than one examination, even two or three, before you are able to feel the tumor. If a tumor is not palpable, the chances are the child is not suffering from pyloric stenosis.

You have already been told that a lot of different diseases will cause vomiting and diarrhea in children, and may give the symptoms and signs of an acute abdomen. If we would only stop to consider the season of the year it happens to be, it would often help us to arrive at a correct diagnosis. We all know that dysentery and typhoid fever are most common in the summer months. Quite often, children will have vomiting and diarrhea and may have signs of an acute abdomen with dysentery and typhoid infection.

We can rule these infections out by doing bacteriologic studies.

Again, the so-called devil's grippe is most common during the summer months, namely July and August, and this disease certainly does give signs and symptoms of an acute abdomen with vomiting and there may be diarrhea.

The best way that I know to rule this disease out is by doing a white blood count. We usually find a leukopenia, or a normal white count with an elevation of temperature, and a relative lymphocytosis.

Then in children, we must also remember that there is such a thing as "green apple" colic. Children can be very sick and have a lot of abdominal pain and diarrhea, because of eating green apples, and during the season when we have green apples, we often see a lot of sickness due to green apple colic.

Again in the fall, or in the late summer, we have to consider the possibility of infantile paralysis and quite often, the first symptoms are vomiting, abdominal pain and rigidity of the spine. If we are immediately aware that we are in the season when we see cases of infantile paralysis, and if there are other cases in the neighborhood, it may help us to arrive at a diagnosis.

Last of all, although not a seasonal incident, I think we should not forget the incident of lead poisoning in children, which causes vomiting and diarrhea and often some abdominal pain. We most certainly should not forget that there is such a condition as acute lead poisoning in infants and children.

PRESIDENT ABBOTT: The meeting is now open for discussion and questions.

DR. SMITH: One question was asked with reference to allergy. I think one condition that is responsible for symptoms in infants and children, even though perhaps it is less common than other possible causes, is improper food, or food disturbances.

The real point which I should like to leave with you, if I may, is that with children or infants who have vomiting and diarrhea, we should not be content with changing the formulas, but should attempt to find the reasons for the symptoms. Very often, we would find these reasons in the four groups that I mentioned previously.

DIURETICS AND WHAT THEY DO*

BY HENRY A. CHRISTIAN, M.D.†

IF diuretics are given to patients with severe acute nephritis, in all probability they will do harm. This judgment is not based on clinical experience, since, for reasons which I shall speak about later, I have never used them in such patients, but it is based on animal experimentation carried out twenty-three years ago by myself and my associates, Drs. Dawson, O'Hare and Walker in which it was found that with a severe experimental nephritis diuretics, such as diuretin, theocin, caffeine, potassium acetate and even water, shortened the lives of the experimental animals, indicating that a diuretic threw an increased burden on the kidney already seriously damaged. Mercurial diuretics were not used in these experiments; modern mercurial diuretics were not at that time in use.

No satisfactory evidence has ever been adduced that diuretics accomplish much more than free the body from accumulated water with its solutes, that is, from oedema, the constituents of which in themselves are not toxic. In severe acute nephritis oedema practically never is a significant cause of discomfort to the patient and so needs no therapy. In patients with acute nephritis with toxic symptoms and a very low urine output diuretics seem to be of little help, and as they may, judged from animal experiment, do harm, it seems wise not to use them in the treatment of such patients. In chronic nephritis with nitrogen retention and toxic manifestations, so-called uremia, clinical experience is that diuretics do not help, and that sometimes here they seem to do injury.

In other words, what we seek to accomplish by diuretics is to remove from the body accumulated fluid that is doing harm by its presence. Moderate oedema, for example of the ankles, is not an indication for the use of diuretics. I do not regard diuretics here as desirable for cosmetic effects. Some of the lady patients might like to have diuretics under those circumstances, but to give them probably is not good practice.

There is a very curious incompatibility between nitrogen retention and the retention of water in the form of oedema, so much so that one can be pretty safe in saying that the patient, who is toxic in this sense, will not be oedematous, unless he has circulatory failure. This point is of practical value to the practitioner who does not have access to a laboratory to determine the blood nitrogen level of his patients.

Symptoms from oedema vary very greatly, depending upon where the fluid is accumulating. Oedema of the brain may be fatal. Hydrothorax may interfere seriously with circulation and respiration. Ascites, unless excessive, is only uncomfortable. Subcutaneous oedema, unless marked, causes few symptoms.

Furthermore, oedema in different locations in the body responds differently to diuretics and particularly with regard to the speed of the disappearance of the oedema. If the oedema is causing serious symptoms, and if drug therapy will remove the oedema but slowly, one should not depend upon diuretics, but should remove the fluid mechanically to relieve the pressure. Again, obviously, it would be foolish to treat severe cerebral oedema by diuretics, because your patient would probably be dead before the diuretic drugs could get started in action, and, of course, death would end their action.

Hydrothorax, which we see so often in people with oedema, responds slowly to diuretic drugs, and on the whole, not very satisfactorily. The needle is wiser than the pill under those circumstances. I believe that hydrothorax in an oedematous patient, particularly one with circulatory difficulty, should be relieved by tapping before you leave the patient on your first visit.

Subcutaneous oedema and ascites are the profitable fields for the use of diuretic drugs. We have available a number of diuretic drugs. In fact, we have available many more than there is any necessity of the practitioner using. The doctor should use only the few with which he is familiar, and it is injudicious for him to use those whose action he does not thoroughly understand.

A good many forms of oedema do not require any diuretic drugs, because treatment of the underlying condition will clear up the oedema without the necessity of using a diuretic drug. For example, every now and then we see a patient with pernicious anemia with a marked degree of oedema. Liver therapy here will take care of the anemia in a relatively short time, and the oedema will fade away without using any diuretic drugs.

Sometimes we see ascites and hydrothorax with pericardial fluid in patients with myxoedema. In such cases thyroid gland substance will clear up the myxoedema symptoms and will also cause elimination of the fluid, and as a rule, no diuretic drug is needed when the oedema is part of a myxoedema. We have certain local

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oedemas which depend upon obstruction of the veins or of the lymphatics or upon local inflammatory conditions. These require treatment other than diuretic drugs and are more surgical than medical in their therapeutic aspects.

The forms of oedema, in which diuretic drugs play their chief rôle of effectiveness, are the forms of oedema that we call renal oedema, the so-called nephrosis syndrome, and circulatory oedema, either cardiac or hepatic. In the former, the oedema mainly is the result of disturbed osmotic relationships between blood plasma and body fluids; in the latter, the oedema results mainly from disturbances in the hydrostatic pressure relationships between circulating blood and extra-capillary pressures. No oedema, however, is so simple as to be reducible to single causative factors, and this plays an influencing part in both mechanism and management.

To remove oedema, we can proceed along two lines. We can do something to change the underlying causative factor, or we can do something to increase the excretion of the retained fluid. For example, in renal oedema, the so-called nephrosis syndrome, we can raise the osmotic tension by an injection of an acacia solution and that will bring about a diuresis. In just the same form of oedema instead of changing the osmotic tension we can increase the output of fluid by using a mercurial diuretic. By direct action on the kidney, this will increase the urine output by decreasing tubular reabsorption.

In cardiac oedema we can combat the cause of oedema by improving the circulation by the use of digitalis. Digitalis, in that sense, is a diuretic. In renal oedema digitalis will have no effect on the fluid; it is in no sense a diuretic for these patients. In the patient with cardiac oedema, in addition to improving the circulation (and we can improve the circulation both generally and in the kidney itself by using digitalis), we can use a diuretic such as some one of the xanthine group, as diuretin or theocin, or some of the combinations of those drugs. We need to remember that some diuretics, such as those of the xanthine group, are effective when given by mouth, while others, such as the mercurials, are ineffective when given by mouth. Obviously, the difference in administration is a determining factor in the selection of the diuretic drug for a given case. By and large it is wiser to use drugs effective by mouth dosage, although sometimes it is more profitable to use drugs that work only after parenteral dosage.

In renal oedema, the so-called nephrosis syndrome, we find that the xanthine diuretics, like theocin and diuretin, are very ineffective, and it is hardly worth while to try them out in this type of patient. On the other hand, the mercurial diuretics are very markedly effective in dealing with the oedema of nephrosis. In modern times we have been slow to use the mercurial diuretics in the patient who had renal disease, because we were fearful that the toxicity of mercury to the kidney would cause increased renal damage. Gradually we have learned that these mercurial diuretics are safe to use in the type of renal disease which expresses itself clinically by the production of oedema.

We have available three mercurial diuretics that are very satisfactory. They are novasurol, salyrgan and mercupurin. Their mercury content increases in the order in which I have named them, and their toxicity decreases in the inverse order to their content in mercury. Mercupurin seems to me, of the group, preferable, but salyrgan is nearly as satisfactory. Both are definitely preferable to novasurol, both in effectiveness and in lessened toxicity.

All of the mercurials must be given parenterally, preferably intravenously, but they may be used intramuscularly, provided the drug is injected deep into the muscle tissues. Novasurol is more irritating than mercupurin or salyrgan; on the whole mercupurin is the least irritating in the muscles. The mercury constituent of mercupurin can be used also in the form of a suppository and thus given is very effective as a diuretic drug. (Since this address was given, it has been found that salyrgan, too, can be prepared so as to be effective in the form of a suppository.) It is not irritating to any appreciable extent to the rectum. It enables the physician to send his patient home with a stock of suppositories with directions how to use them, thus obviating the necessity of the physician's presence to give an injection intravenously or intramuscularly. Like other drugs given by rectum the dose by rectum is about five times the dose given by mouth, intravenously or intramuscularly.

Acacia solutions are effective in renal oedema, but their use is cumbersome, and it is difficult to obtain acacia solutions that do not cause a reaction.

In regard to circulatory oedema, as I have already said, digitalis is effective and should be used first. The xanthine diuretics that can be given by mouth are really very effective. We have gotten out of the habit of using them, but our therapy would be more easily carried out, if we would return to their use in cardiac cases where the oedema is in excess of that satisfactorily taken care of by the digitalis. If the xanthine diuretics do not work, then one can use the mercurials in the way I have just referred to. They are very effective.

We now come to hepatic oedema, ascites, usually from cirrhosis of the liver. Diuretics of a certain type here produce very good diuresis,

but they rarely do more than prolong the interval between the necessity of tapping the abdomen. However, that is worthwhile to the patient. Consequently, in hepatic oedema try out diuretics, to see their effectiveness. As a rule, mercurial diuretics here will be more effective than the xanthine group.

In all of these three forms of oedema, urea in large doses by mouth, sixty to ninety grams per day, may cause a satisfactory diuresis, but many patients find it difficult or impossible to take urea, even if dissolved in fruit juice. It frequently causes marked nausea, often vomiting, and hence its field of usefulness is very limited.

It should be remembered in using diuretics that one may bring about too much dehydration of the body, and that as a result the patient may get into difficulty because of this. It is quite possible to have some of the tissues of the body dehydrated to the point of producing discomfort and symptoms, when other parts of the body are definitely water-logged. Too great a restriction of fluids during diuresis will do harm, and, strikingly often, a little fluid added to the intake will increase the urine output and even promote a large diuresis.

The cirrhotic patient with a belly full of fluid and with emaciated, dehydrated tissues is sometimes made worse by a diuretic drug and made better by a good drink of water or orange juice. The proper combination, or the proper amount of fluid intake, in relation to diuretic drugs should always be kept in mind.

Practically all of these diuretic drugs also remove from the body large amounts of sodium chloride. The patient with sodium chloride deficiency in the body already is in trouble. A little judicious adding of salt to the diet of the oedematous patient often will greatly improve the general clinical condition of the patient, and not infrequently a judicious sprinkling of salt on the food will increase the effectiveness of your diuretic drug.

So, in your enthusiasm for relieving the body of oedema, do not forget the discomforts and the disabilities of dehydration and deficient sodium chloride in the body.

DISCUSSION

PRESIDENT ABBOTT: Discussion of this paper will be opened by Dr. Bruce Snow, of Manchester, New Hampshire.

DR. BRUCE SNOW: First of all we want to thank Dr. Christian for his kindness in coming here to speak to us today, and I also want to call attention to the thoroughness with which he has covered the subject. Almost twenty years ago when, as a student in medical school, I was listening to Dr. Christian speak to us on kidneys, hearts, digitalis and other fascinating subjects, I was often amazed at the extensive knowledge shown and this feeling is still with me today.

When first asked to discuss this paper, I hesitated to do so because I knew that after Dr. Christian had spoken there would be very little for me to add. This has been amply demonstrated, but I would like to add a few observations of my own which may be, to some extent, a repetition of what has been said.

I have always been deeply impressed by the many limitations in the usefulness of diuretics. We must bear in mind that:—

1. They are not to be used in acute nephritis.
2. They are not to be used in the milder forms of non-disabling oedema.
3. They are not of much use in oedema of the body cavities.
4. They are not of value in removing toxic products from the blood in nephritis, uremia and other toxemias.
5. They are of little value in the obstructional and rarer forms of oedema.

With the elimination of the above conditions there remain only two main causes for oedema: nephrosis and cardiac insufficiency. As Dr. Christian has so fully brought out, the oedema of nephrosis is helped not at all by the xanthine diuretics and only partially, though to a valuable extent, by the mercury group, whereas cardiac oedema is greatly improved by rest and digitalis with the addition, if necessary, of the xanthine and mercurial diuretics. The oedema of nephrosis is helped by the mercurials alone, while cardiac oedema is aided by all types of diuretics and it is in the latter group with combined treatment that we receive our most definite results.

I think it is well to remember, however, that practically and clinically there is often no sharp dividing line between cardiac and nephrotic oedema —that many oedemas are mixtures of the two types. In the late stages of chronic glomerulonephritis we often see patients develop hypertension and cardiac damage so that the ensuing oedema is to a large extent due to the heart rather than to the kidneys; such a type may receive great help. The practical application of this is that, in all severe oedemas which are not certainly of obstructive origin, it is wise to watch cautiously the successive effects of digitalis, theobromine and salyrgan for many of these oedemas will prove to have some cardiac origin and will gain marked relief if properly treated.

(Dr. Walter F. Taylor of Keene, New Hampshire then discussed the paper and corroborated in every detail the statements made by Dr. Snow.)

PRESIDENT ABBOTT: If there is no further discussion of this paper, I shall ask Dr. Christian to close the discussion.

DR. HENRY A. CHRISTIAN: There is just one thing that I do not think I made quite clear. That is with regard to fluid in the abdominal cavity, ascites. If it is due to a blocking of the liver from cirrhosis, then you do not have any very striking prolonged influence from the use of diuretics; you can produce the diuresis, but the fluid rapidly reaccumulates. On the other hand, if the ascites is due to circulatory diseases, that is, to cardiac insufficiency, digitalis and diuretic drugs, singly or in combination, have a very strikingly rapid effect on the removal of the fluid, and, when it is removed, it will not recur until circulation fails again. So, you must distinguish between ascites from cirrhosis of the liver and ascites from cardiac failure, the latter being particularly amenable to diuretic drugs.

MEDICAL PROGRESS

PROGRESS IN GASTRO-ENTEROLOGY FOR 1935

BY EDWARD S. EMERY, JR., M.D.*

FOR a review of this kind no attempt has been made to abstract every article pertaining to the gastrointestinal tract. To do so would make a summary of this kind unnecessarily bulky, as many articles are of a statistical nature, which although useful, do not contribute anything new to our knowledge of the subject, and some are of a theoretical nature which would be of interest to only a comparatively few readers. An attempt has been made to review those articles which would seem to be of interest to the general reader. No claim is made for completeness, and doubtless certain articles were missed which could have been profitably included.

TOPICS BEARING ON THE GENERAL SUBJECT

Thomas Brown has written an article which he entitles, "The Philosophic, Clinical, and Retrospective Discussion of Certain Major Problems in the Digestive Field" which is unusually well done; shows a great deal of common sense, and should be read by everyone, whether or not he is particularly interested in the gastrointestinal tract.

Alvarez has pointed out what all physicians should remember — that many elderly persons complaining of acute indigestion may have suffered from an unrecognized cerebral hemorrhage. He believes that after a "stroke" the digestive tract may become spastic, much as an injured arm or leg is spastic; or, "storms" may go down the vagus nerve, much as they do in migraine. He gives brief histories of five patients who suffered from this type of accident with resulting digestive symptoms. In some of these cases, the true cause of the condition was not recognized for some time.

De Beer et al. have studied the composition of the intestinal secretion by making isolated loops in dogs at various levels. After allowing a sufficient time for complete healing, they have been able to follow the secretions in the unanesthetized animals. They found that the rate of secretion is greater in the jejunal and ileal loops than in the colon. The concentration of sodium, potassium and calcium is relatively constant in all the secretions, whereas, the bicarbonate and chloride ions show marked reciprocal variations. The juices in the lower ileum and colon which are known to be more alkaline than the jejunal juices, show a relatively high content of bicarbonate, and a relatively low amount of chloride, which was the reverse in the secretion from the upper intestinal tract. They found that increasing the blood chloride by the intravenous injection of sodium chloride caused a similar change in the jejunal and ileal secretions. On the other hand, intravenous injection of sodium carbonate caused no characteristic variations in the jejunal loop.

Herrin has studied the chemical changes in the blood and in the intestinal secretions produced by the loss of intestinal juices. This article is worth reading by anyone interested in this subject.

Brown, T. R.: Philosophic, clinical, and retrospective discussion of certain major problems in the digestive field. Am. J. Digest. Dis. & Nutrition **2**:391 (Sept.) 1935.
Alvarez, W. C.: Unrecognized "strokes" and the gastro-enterologist. Am. J. Digest. Dis. & Nutrition **2**:90 (April) 1935.
de Beer, E. J.; Johnston, C. G., and Wilson, D. W.: The composition of intestinal secretions. J. Biol. Chem. **108**:113 (Jan.) 1935.
Herrin, R. C.: Chemical changes in blood and intestinal juice produced by the loss of intestinal juice. J. Biol. Chem. **108**:547 (Feb.) 1935.

Vitamins and the Digestive Tract

Altschule reports microscopic evidence of a deficiency of vitamin A in six of eleven infants who suffered from protracted jaundice as the result of a congenital atresia of the bile ducts, in spite of the fact that the children had received a normal diet. The findings were thought to result from a deficient absorption of vitamin A because of lack of bile.

Loewenthal, from a study of 1,000 school children in Teso, Ugandi, concludes that the diarrhea and dysenteries rather commonly encountered there are not a manifestation of vitamin A deficiency, but are due to some other deficiency, the nature of which has not yet been recognized.

Sure et al. have studied the activity of pancreatic enzymes in vitamin B avitaminosis. There was a marked reduction in the efficiency of the pancreatic lypase and esterase, but no disturbance in the tryptic or ereptic activity.

Steinbach and Rosenblatt conclude that a high vitamin diet has no beneficial effect on intestinal tuberculosis.

Altschule, M. D.: Vitamin A deficiency in spite of adequate diet in congenital atresia of bile ducts and jaundice. Arch. Path. **20**:845 (Dec.) 1935.
Loewenthal, L. J. A.: The manifestations of vitamin A deficiency in man. Ann. Trop. Med. **29**:407 (Dec.) 1935.
Sure, B.; Kik, M. C., and Buchanan, K. S.: Enzymatic efficiency in avitaminosis. J. Biol. Chem. **108**:27 (Jan.) 1935.
Steinbach, M. M., and Rosenblatt, M. B.: Vitamine therapy in intestinal tuberculosis. Am. Rev. Tuberc. **31**:35 (Jan.) 1935.

X-Rays and the Digestive Tract

Holmes and Schatzki describe the examination of the mucosa by means of the x-ray and give

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illustrations that demonstrate different types of relief patterns. They discuss the possible advantages of this type of study over the usual method.

Pendergrass and Comroe report on the roentgenologic appearance of the gastrointestinal tract in a case of chronic idiopathic adult tetany. The stomach was found to be hypertonic and showed a definite hypermotility in spite of a hypoperistalsis. The duodenum was widely dilated, the small intestine was moderately dilated, and showed considerable stasis. Little pools of barium were scattered throughout the small intestine. The valvulae conniventes were apparently hypotonic and could not be visualized. The colon appeared to be normal. (Other articles have appeared from time to time suggesting a definite change in the roentgenologic appearance of the gastrointestinal tract in such conditions as hyperparathyroidism in which the calcium metabolism is affected. The findings, as reported in these different accounts, do not agree entirely. It is difficult to say to what degree the changes depend upon the metabolic disorder. However, it seems probable that disturbances in the physiology of the intestinal tract do occur, and further observations should be made on these cases.)

Jones reports seven cases of cicatricial stenosis which occurred in the intestinal tract months or years after intensive roentgenotherapy for uterine malignancy. (This is a complication which all of us should keep in mind.)

Holmes, G. W., and Schatzki, R.: Examination of the mucosal relief as a diagnostic aid in diseases of the gastrointestinal tract. Am. J. Roentgenol. **34**:145 (Aug.) 1935.
Pendergrass, E. P., and Comroe, B. I.: Roentgen study of gastro-intestinal tract in chronic idiopathic adult tetany. Am. J. Roentgenol. **33**:647 (May) 1935.
Jones, T. E.: Intestinal complications resulting from prolonged radium and x-ray irradiation for malignant conditions of pelvic organs. Am. J. Obst. & Gynec. **29**:309 (March) 1935.

ESOPHAGUS

Knight has studied the relation of the extrinsic nerves to the functional activity of the esophagus. Working with cats, he found that by cutting the fibers of the vagus which supplies the lower end of the esophagus, the animals developed a condition apparently analogous to cardiospasm. By removing the celiac plexus, he was able to restore the esophagus to a normal state. Furthermore, he found that if the celiac plexus is removed before the vagal fibers are cut the animals show no tendency to develop cardiospasm. (This work supports the idea held particularly by English students that cardiospasm depends upon a faulty innervation of the lower esophagus.)

Rogers has relieved a patient with dysphagia and anemia (the so-called Plummer-Vincent syndrome) of two and a half years' duration, by bilateral removal of the superior cervical ganglions. It left the patient with a bilateral Horner's syndrome. The author does not advocate it for all patients, but feels that it is worth trying on severe intractable cases.

Otell and Coe discuss dysphagia from the roentgenologic point of view and give a list of various conditions which may be responsible for this symptom. They separate the causes of dysphagia as follows:

Group 1: (most common)	Foreign bodies
	Achalasia of the cardia (cardiospasm)
	New growths
	Esophageal orifice hernia
Group 2: (less common)	Compression stenosis
	Diverticulum and pharyngeal pouches
	Congenital atresia
	Paralysis of the esophagus
	Cicatricial stenosis
	Esophageal varices
	Dysphagia of anemic women
Group 3: (rare)	Benign tumor
	Peptic ulcer of the esophagus
	Thoracic stomach
	Syphilis
	Tuberculosis
	Esophagitis
	Mycotic stenosis
	Dysphagia associated with gastric lesions
	Herpes and urticaria
	Angioneurotic edema
	Serum sickness
	Spasm of the esophagus
	Atony of the esophagus
	Globus hystericus and hysteria

MacMillan has reviewed 1,600 cases in which the patient sought relief from dysphagia. Foreign bodies were found in 181 cases. Malignancy was the complaint in 350, cardiospasm in 135, and narrowing appeared in the upper portion of the esophagus in 96. A web-like formation was present in 114 patients. Extrinsic conditions were responsible for the dysphagia in 31 patients. Strictures as the result of burns to the esophagus were found in 40. Forty-six patients suffered from paralysis of the esophagus due to such conditions as poliomyelitis, cerebral hemorrhage, brain tumor, fracture of the skull, and Parkinson's disease. Pharyngeal diverticula were found in 32 cases.

Bartels discusses acute ulcerative esophagitis. He reports on 82 cases, and states that they can be divided into the following groups:

1. Pseudomembranous ulceration.
2. Simple complication with slight inflammation.
3. Hemorrhagic ulcer.
4. Phlegmon of the entire wall.

Patterson tells of the case of a 67 year old housewife who spit up a cast of the entire esophagus. She had been complaining of symptoms for several weeks and after this episode,

her trouble gradually subsided. He was not able to determine the etiology, although the patient was known to be addicted to alcohol. The author gives a good review of the literature which will be useful for anyone looking up this subject.

Glass and Freeman report two instances of spontaneous rupture of the esophagus in patients who were suffering from "paretic seizure." Both of these patients died shortly from hemorrhage, and autopsy revealed periarteritis in the esophageal wall. The ruptures occurred during the periods of vomiting, and the authors suggest that the regurgitation of autolytic gastric contents eroded the lining of the esophagus which had been damaged by the syphilitic reaction.

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Rogers, L.: The treatment of spasmodic dysphagia by sympathetic denervation. Brit. J. Surg. **22**:829 (April) 1935.
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MacMillan, A. S.: Statistical study of diseases of the oesophagus. Surg. Gynec. & Obst. **60**:394 (Feb.) 1935.
Bartels, E. C.: Acute ulcerative esophagitis; pathologic and clinical study of 82 cases observed at necropsy. Arch. Path. **20**:369 (Sept.) 1935.
Patterson, T. C.: A simple superficial oesophageal cast (oesophagitis exfoliativa: oesophagitis dissecans superficialis). J. Path. & Bact. **40**:559 (May) 1935.
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STOMACH

Physiology of the Stomach

Day and Webster have investigated the autoregulation of the gastric secretions. This work was done on two dogs, on which was performed an esophagotomy and gastric and duodenal fistulas. The stomach was finally disconnected from the duodenum. The obstructed animals were fed in the usual manner with the food mixture described by Scott and Ivy, which was introduced into the duodenum through the fistula. In certain experiments, solutions of gastric juice of known acidity were introduced into the duodenum. These workers found that the introduction into the duodenum of 0.25 per cent hydrochloric acid or of gastric juice diluted two to four times, inhibits the gastric secretion stimulated:

1. By the parasympathetic nervous system.
2. By the presence in the intestines of food substances, or the products of their digestion.

The same effect was produced by the acid in some cases of hypersecretion of the gastric glands. They advance the theory that the passage of the acid chyme from the stomach into the duodenum causes gastric secretion to diminish, this being an important factor in regulating the activity of the gastric glands.

Kabat et al. report that electrical stimulation of the lateral hypothalamic area causes cessation of peristalsis and loss of tone in the stomach and small intestine. However, stimulation of the thalamus, internal capsule, anterior commissure, septum pellucidum and infundibular stalk, produced no change in the gastrointestinal motility.

Hellebrandt has found that the recurrent hunger cycles of the human fasting stomach are associated with fluctuations in the acidity of the gastric content. These two phases of gastric function; namely, motility and secretion, increase and subside in unison. Hellebrandt, this time with others, has studied the effect of acute anoxemia on the gastric functions. Acute anoxemia seems to have little inhibiting influence upon the secretion of hydrochloric acid or the other functions of the stomach. They believe that the suppression of gastric activity after exercise cannot be explained solely by anoxemia.

Bowman et al. have injected various amino-acids intravenously: namely, glycine, cystine, leucine, isoleucine, tyrosine, proline, tryptophane, lysine, arginine, and histidine. In the forty-three experiments that were performed on three dogs, the authors were not able to influence gastric motility, and hence conclude that the concentration of amino-acids in the blood is of no importance from this point of view.

Wilhelmj et al. have studied the secretions of the stomach and duodenum. They found that the neutral chloride concentrations of the non-acid secretions of the stomach and mixed duodenal secretions were approximately the same, the average values ranging from 358 to 357 mg. in 100 cc. They found also, that only about one-third of the neutral chloride represents hydrochloric acid that has been neutralized. Therefore, they believe, that about two-thirds of the neutral chloride found in these juices are secreted as such.

Heckmann points out that under normal conditions the fundus of the stomach is in close contact with the diaphragm. Failure to find this normal relationship may be the result of ascites, perigastric adhesions, or carcinoma of the colon, working its way between the stomach and the diaphragm. He suggests that this portion of the stomach is important in regulating the pressure exerted on the gastric content.

Wayne and Graybiel conclude that eating increases the tendency to anginal pain in patients with heart disease, by increasing the amount of work which the heart has to do. They found that an average reduction of 25 per cent in exercise tolerance occurred after a heavy meal, but these same patients were not affected if the stomach was distended sufficiently to change the position of the heart. However, they found other cases in which both food and inflation of the stomach with air would bring on an attack.

They believe that this result has to be explained by some kind of reflex action.

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Heckmann, K.: Zur Morphologie und funktionellen Bedeutung der Magenblase. Med. Klin. **31**:611 (May 10) 1935.
Wayne, E. J., and Graybiel, A.: Observations on the effect of food, gastric distension, external temperature, and repeated exercise on angina of effort, with a note on angina sine dolore. Clin. Sc. **1**:287 (Nov.) 1934.

Diseases of the Stomach

Sagel describes two cases which came to autopsy as the result of benign strictures of the stomach and esophagus. The first was a patient, aged 42, who died thirty-eight years after swallowing lye. The second occurred four weeks after drinking moonshine. The lower esophagus in each case was scarred. The lesser curvature of the stomach was essentially normal, but the pylorus and duodenum were damaged. Thick scar tissue was present on both sides of the pylorus. (The cases are interesting in showing how irritating liquids are held up in the cardia and again in the pylorus. It is also interesting that the duodenum was involved for only a short distance beyond the pylorus.)

Pendergrass and Andrews point out that by means of the fluoroscope one can usually make a diagnosis of a prolapse through the pylorus of gastric mucosa, or a gastric polyp. Errors are not infrequently made by mistaking congenital mesenteric membranes, redundant normal membranes, inflammatory adhesions, and hypertrophic pyloric stenosis for these conditions. The authors have had eight cases, in five of which they diagnosed a polyp, and in three a prolapse of the gastric mucosa. In reality, a polyp was present in four and a prolapsed mucosa in four.

Levin believes that the intraperitoneal insertion of radon plus the operation of gastroenterostomy is at times of value in carcinoma of the pyloric end of the stomach. He reports satisfactory results in six cases which he observed when director of the New York City Cancer Clinic.

Gray and Melnick have determined the gastric acidity in fifty patients with pulmonary tuberculosis who were between the ages of twenty and thirty years. He believes that the disease produces hypo-acidity in one-third of the patients, and an anacidity in one-fourth of the patients in from one to four years after symptoms begin. Also, he states that as the acid decreases, tubercle bacilli are found more frequently in the stomach contents.

Edwards believes that gastroscopy with the Wolf-Schindler flexible gastroscope is of great assistance in studying patients with suspected carcinoma, chronic gastritis or jejunal ulcers following gastro-enterostomy.

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Gray, I., and Melnick, J.: Gastric acidity in pulmonary tuberculosis; study of 50 patients between ages of 20 and 30 years. Am. Rev. Tuberc. **31**:460 (April) 1935.
Edwards, H. C.: The value of gastroscopy. Lancet **2**:1161 (Nov. 23) 1935.

Diaphragmatic Hernia

Numerous papers have been written on the subject of diaphragmatic hernia of which only two have been abstracted. Dunhill has written a statistical report of twenty-five cases. Eleven had an esophagus of normal length, fourteen had a congenitally short esophagus and the stomach was found in the thorax. Sites of hernial orifice were as follows:

Retrosternal	1
Dome, left diaphragm	2
Costovertebral region	3

Five were in the region of the esophagus, of which three were due to maldevelopment of the crura, and two to a para-esophageal hiatus which had become dilated.

Einhorn et al. report symptoms of cardiospasm which were the result of a diaphragmatic gastric hernia. The patient had symptoms of dysphagia, and a bougie met with resistance about fifteen inches from the teeth. The true nature of the disease was missed in the first x-ray examination, but a second one revealed the diaphragmatic hernia. The diaphragm was surgically repaired, with complete relief up to the time of the report.

McGill has given a good synopsis of congenital pyloric stenosis and discusses the theoretical causes of this interesting condition.

Dunhill, T.: Diaphragmatic hernia. Brit. J. Surg. **22**:475 (Jan.) 1934-35.
Einhorn, M.; Stetten, D., and Stewart, W. H.: Report of case of cardiospasm due to diaphragmatic gastric hernia with ulcer; operative procedure. J. Thoracic Surg. **4**:310 (Feb.) 1935.
McGill, C. F.: Congenital pyloric obstruction. New Eng. J. Med. **213**:567 (Sept. 19) 1935.

PEPTIC ULCER

Etiology

More experimental work has been done in 1935 to support the idea that the gastric acidity has a close association with peptic ulcer.

Harper isolated loops of the jejunum and stitched the distal end of this loop to an isolated gastric pouch and then brought the proximal end out through a stab wound in the abdomen. Reversal of the loop served to keep the mucous membrane constantly bathed with unneutralized hydrochloric acid. Ulcers developed near the anastomosis, and Harper believes that three factors were responsible for the development of the ulcerations:

1. The chemical action of the hydrochloric acid seemed necessary because ulcers did not develop if rapid drainage was permitted through the loop, or if the contents of the pouch were neutralized.
2. A mechanical factor was thought to be of importance because the ulcer developed at the point of mechanical interference with the outflow of juice.
3. Susceptibility of the mucosa also plays a rôle, because, as McMaster has previously reported, there was a greater tendency for ulcer to develop when one used loops from lower levels of the intestine.

(This work confirms the ideas which Williamson has already suggested.)

By making a Pavlov pouch in the duodenum of dogs, Stelzer was able to demonstrate that a high acid would produce ulcers in the duodenum. The pouch was made in the infrapapillary portion of the duodenum. Later, he made an anastomosis between this pouch and the stomach. The animals were given daily hypodermic injections of histidine for from six to eight weeks, at the end of which time the animals were killed. Typical ulcers were found in this area. It is felt by the authors that nervous influences, muscular spasms, and circulatory disturbances can be ruled out, and damage to the mucous membranes must be ascribed to the peptic action of the gastric juice.

Wu has studied the reaction of the jejunal contents after "surgical drainage of the duodenum". The jejunal contents were found to have a higher acidity, and there was a greater fluctuation in this acidity following the operation of "surgical drainage of the duodenum". Under normal conditions, he did not find that any change in acidity occurred with the development of ulcer. (This is opposed to some recent work by Ivy.)

McMaster has performed a rather interesting experiment, the results of which are hard to interpret. He produced an anastomosis between the duodenum and colon on twenty-seven dogs which survived. In sixteen of the animals the jejunum and ileum were left as a blind stump, the proximal end having been closed. Thirteen of these animals developed ulcer, three of which perforated, an incidence of 65 per cent. In eleven of the dogs the jejunum and ileum were resected and only two of these animals developed an ulcer, an incidence of 18 per cent. It is difficult to say whether these findings are the result of anything more than chance. Further studies along these lines will be worthwhile.

Graves endeavored to compare the efficacy of bile and pancreatic secretions in the prevention of the development of experimental peptic ulcer. If Williamson's operation of "surgical drainage of the duodenum" was performed on dogs and the duodenal contents drained into the proximal portion of the stomach, no ulcer formed in the jejunum which had been anastomosed to the pylorus. An ulcer formed if "surgical drainage of the duodenum" was done and either the bile or pancreatic juice alone was drained into the jejunum. However, either of these secretions was sufficient to prevent an ulcer forming in the duodenum.

Blanck has studied the effect of exclusion of bile on the development of ulcer. He effected a complete external drainage of bile in eight dogs. Five were fed only a laboratory stock diet, while the remaining three were given the same diet plus the bile that had been collected externally. The dogs without bile feedings developed not only peptic ulceration, but the associated pathologic changes of gastritis, duodenitis, and jejunitis. By incorporating fresh bile with the feedings, both peptic ulcers and the associated pathologic changes could be prevented. (It is unfortunate that Blanck did not go a step farther and try feeding bile after an ulcer had developed in order to determine if it were possible to cause healing after the ulcer had once formed. Experiments such as these show pretty conclusively, that peptic ulcer depends upon at least two factors:

1. The corrosive effect of hydrochloric acid.
2. The resistance of mucous membranes to the corrosion.

Unfortunately, no method has been developed, as yet, to determine which of these factors is of importance in the individual patient.)

Hoff and Sheehan have investigated the effect of damaging the hypothalamus on the intestinal tract of monkeys. Five of sixteen monkeys showed hemorrhagic erosions of the mucosa of the stomach following injury to the hypothalamus. These lesions were limited to the stomach. Three of the five showed dilatation and atony. According to the authors, these findings were not conclusive of sympathetic activity. All of the five which showed the hemorrhagic erosions were shown to have received an injury to the tuberal nuclei.

McLaughlin gives further evidence that partial bilateral destruction of the adrenals produces ulceration in the intestinal tract, especially the duodenum. He was not able to prevent this by sectioning the vagi on both sides.

Within the last few years it has become evident that the incidence of peptic ulcer is high in patients suffering from polycythemia vera. This has stimulated workers to study the blood of patients with ulcer to discover if there is any evidence of polycythemia.

Ugelli has examined the blood of ninety-two patients suffering from peptic ulcer, and reports that erythrocytosis was proved in 74 per cent of the patients with duodenal ulcers, in 40 per cent of the patients with gastric ulcers, and in 40 per cent of a group that had gastric resections performed from seven months to three years before the examination.

From Otto's study of several hundred patients with ulcer, he believes that erythrocytosis exists in a large number. (However, from some studies which the reviewer has made recently, he does not believe there is any reason to feel that the condition of peptic ulcer, as ordinarily seen, is analogous to polycythemia. It seems probable that the high incidence of ulceration in patients with polycythemia depends upon the vascular changes which are known to exist in this disease.)

Fetterman has studied thirty stomachs with ulceration for evidence of vascular lesions. Thirteen of these stomachs came from patients with duodenal ulcer and seven with gastric ulcer. Seven showed marked internal thickening of the arteries in the vicinity of the ulcer and at some distance from the ulcer. The thickening occurred in isolated areas of the stomach and which he believes were of an infective origin.

Morrison and Feldman have fed to dogs large quantities of bacteria mixed with the food. They were unable to demonstrate that this had any effect on the animals even though ulcers were produced by the injection of one per cent hydrochloric acid. The ulcers healed as rapidly as those in a control group to which no bacteria were given.

Robinson has studied patients with peptic ulcer from the point of view of racial selectivity, anthropometric selectivity, personality selectivity, sex selectivity, site selectivity. He states that there is good reason to feel that the disease can be placed on a psychogenic basis. He believes from this investigation, that the condition occurs only among susceptible individuals of the white race, usually of the long, thin type who are given to worry and nervous instability. These individuals with their fears and anxieties develop an imbalance of the vegetative nervous system, which results in vascular spasm, thrombosis, induration, ischemia and finally necrosis and ulceration.

Winkelstein has made 169 studies of the nocturnal gastric secretion on 62 cases of duodenal ulcer, 29 cases of gastric ulcer, 17 cases of gastric carcinoma, 32 cases of partial gastrectomy, 5 cases with gastro-enterostomy, and 20 controls. He found that where normal individuals have little or no free hydrochloric acid during the night, the patients with duodenal ulcer, gastric and jejunal ulcer have a high nocturnal curve, and a high titre of free acid. Patients with gastric carcinoma show a low acidity with increased mucus and blood. The author was unable to control the hypersecretion and hyperchlorhydria by alkalies, olive oil, atropine, or by aspiration. The best method of controlling the secretion is by a milk drip or surgically, by means of a partial gastrectomy.

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Winkelstein, A.: 169 studies in gastric secretion during night. Am. J. Digest. Dis. & Nutrition **1**:778 (Jan.) 1935.

Treatment of Peptic Ulcer

Florey and Harding have made a careful and valuable study of the rate of healing of mechanical lesions produced in the duodenum of the cat. They opened the duodenum and snipped off the mucosa down to the circular muscle. They describe the various changes that occur in the process of healing, which required 180 days before the duodenum became entirely normal. (This supports the claims of those clinicians who believe that treatment of ulcer should be continued intensively for long periods of time. It is generally recognized that peptic ulcer is a chronic disease for which there is no cure, and for which treatment should stress preventive measures. The work of Florey and Harding suggests that intensive treatment for a period of at least six months is indicated if the best results are to be obtained.)

Recently much interest has been attached to the treatment of peptic ulcer with histidine monohydrochloride.

Rafsky reports that this treatment produced a marked decrease in acidity, at times to the point of anacidity in one-fourth of his cases. Symptomatic relief was obtained in 73.3 per

cent. The patients who did not respond to treatment were those with a penetrating duodenal ulcer and a niche.

Smith has tried the histidine treatment on twelve cases. He writes quite enthusiastically. The first symptom to disappear was discomfort. Vomiting when present was almost immediately controlled, and there was a speedy return to a desire for food in those patients whose appetite was poor.

Eads has treated thirty-five patients. Seventeen per cent showed immediate clinical and x-ray evidence of healing. Eight cases, or 27.8 per cent, improved more slowly, but nevertheless definitely. Nine cases, or 25.7 per cent showed some clinical improvement, but no change by the x-ray. Thirty-four per cent were unimproved. (However, an improvement in 70 per cent of the cases can be obtained by almost any type of treatment, so that the results to date do not indicate that a lack of histidine is the cause of all cases of ulcer. One must remember that this treatment must of necessity be a palliative one. If the disease of ulcer depends upon a deficiency in histidine, continuous treatment with this amino-acid will be necessary.)

Seely and Zollinger have resected the fundus of the stomach in four dogs leaving a tube of about 3 cm. in diameter from the esophageal opening to within 6 cm. of the pylorus. An immediate drop in the free and total acidities occurred, but these returned to preoperative levels by the end of eight months. (This confirms the work of other investigators, that a permanent lowering of the gastric acidity cannot be obtained by partial removal of the stomach.)

Rivers has treated patients with an extract from the duodenum in addition to the use of some alkali and belladonna. No striking improvement in the results of treatment have been obtained.

Gordon-Taylor believes that surgery is not used enough in cases of bleeding ulcer. He discusses surgical prophylaxis by the use of a continuous slow drip transfusion of blood. He believes that immediate operation is needed in cases with "terrific" bleeding, and where there is concomitant bleeding plus perforation. He believes in an early operation for those patients in whom drip transfusion fails to control the bleeding. He advises operation for the deep penetrating ulcer, whether the bleeding is severe or slight, and this is true also for the cases with a long, definite ulcer history, stenosis, mid-gastric narrowing, and a second hemorrhage occurring within a year.

In 1931 Meulengracht started to feed patients with a bleeding ulcer. Previous to this he had treated them by the old method of starvation during the bleeding period. Since 1931, he has given an adequate diet of puréed foods and a powder consisting of bicarbonate of soda, magnesium subcarbonate aa. 15.0 grams plus extract hyoscyamus 2.0 grams. A teaspoonful of this mixture is given three times a day and in addition he gives 0.5 grams of "ferrilacta" t.d.s. Of 251 cases, three have died. Under the former method of treatment, melena persisted for an average of 13.4 days, with an average of 4.5 defecations. Under the present method, the period of melena dropped to 10.2 days with an average of 4.8 defecations. He has observed that death from a hemorrhage due to an ulcer, occurs on the average, eight days after the hemorrhage begins.

Meyer et al. have studied the action of oil of peppermint on the secretion and motility of the stomach in patients with peptic ulcer. They find that it tends to decrease the secretion of acid and that the healing time is shortened. Although they have shown that this effect is not due to the menthol contained in the oil, they have not yet discovered just what is the cause of this action. Experiments performed on dogs with gastric pouches suggest that the oil of peppermint affects the mucosa directly.

Florey, H. W., and Harding, H. E.: The healing of artificial defects of duodenal mucosa. J. Path. & Bact. **40**:211 (March) 1935.

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Smith, D.: Histidine treatment of peptic ulcer of lesser curvature, with note on 12 cases. Brit. M. J. **2**:154 (July 27) 1935.

Eads, J. T.: Histidine in treatment of peptic ulcer: preliminary report. Am. J. Digest. Dis. & Nutrition **2**:426 (Sept.) 1935.

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Rivers, A. B.: Use of duodenal extract as adjuvant in treatment of benign peptic lesions: report of 8 cases. Am. J. Digest. Dis. & Nutrition **2**:189 (May) 1935.

Gordon-Taylor, G.: The attitude of surgery to haematemesis. Lancet **2**:811 (Oct. 12) 1935.

Meulengracht, E.: Treatment of haematemesis and melaena with food. Lancet **2**:1220 (Nov. 30) 1935.

Meyer, J.; Scheman, L., and Necheles, H.: Action of oil of peppermint on secretion and motility of stomach in man. Arch. Int. Med. **56**:88 (July) 1935.

Further Statistics on Peptic Ulcer.

Wright reports on a collective inquiry by the Fellows of the Association of Surgeons into gastrojejunal ulceration. Following operation for duodenal ulcer, there was an incidence of 4.14 per cent of proved jejunal ulcerations and 8.63 per cent of proved plus suspected ulcers. In the case of gastric ulcer there was an incidence of 2.32 per cent of proved jejunal ulcer and 4.48 per cent of proved plus suspected ulcers. The onset of symptoms came within two years in 62 per cent of the cases. In 285 cases the ulcer was at the anastomosis, and in 99, just beyond.

Christiansen has studied 21 fatal cases of massive hemorrhage upon whom autopsies were performed in 16. Twelve revealed erosion of fairly large arteries. The time that elapsed from the beginning of the hemorrhage varied from 3 to 30 days. Because it is hardly possible that a

patient may live for any number of hours with a continual arterial hemorrhage, it seems reasonable to conclude that the hemorrhage must have stopped some time before death, and consequently could not be the cause of death in the strictest sense of the term. The writer suggests that the cause of death in these cases may have been uremia.

A study of the death rate in New York City from 1900 to 1933 shows the interesting fact that the death rate per hundred thousand of the population, from peptic ulcer, has risen from 3.69 in 1900, to 6.77 in 1933, having reached its peak, 7.51 in 1928. During this period there has been a decline in the death rate among females. In the beginning of the century it was nearly 1 to 1 for the sexes, now the rate is approximately 5 males to 1 female.

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Christiansen, T.: Uraemia as cause of death in massive haemorrhage from peptic ulcer. Acta med. Scandinav. **85**:333, 1935.
"Some puzzling facts on peptic ulcer." Quart. Bull. City of New York. Department of Health. **2**:84, 1934.

DUODENUM

Several patients are reported with unusual conditions of the duodenum.

Bonar observed a case of congenital atresia of the duodenum in a child 13 months of age, who died following a gastro-enterostomy. At operation there was a stricture of the duodenum at the point over which the mesenteric vessels cross, but this was not due to pressure from the vessels.

Nicholson studied a man 66 years of age whose complaint was jaundice. There was a mass in the right upper quadrant and ascites. X-ray studies were negative except for a diverticulum of the duodenum. Later, an autopsy revealed that the jaundice was the result of the diverticulum. The pouch was large enough for its tip to become adherent to the side of the duodenum, forming an angle at the base. This angle acted as a valve for food which entered the pouch. When full, the diverticulum pressed against the common duct.

Ockerblad and Gonzales report 1 case of their own, and review 25 others in the literature, of a duodenal fistula resulting from a nephrectomy for a perinephritic abscess. There are two main causes for this condition. One is trauma to the unperitonealized duodenum with forceps. The second is the result of an abscess eroding into the duodenum. There were 12 deaths from this condition in the 75 cases previously reported in the literature.

Lawson gives a rather complete study of duodenal diverticula. By means of the x-ray he has detected 36 instances of this condition in 2,250 consecutive examinations of this organ. Fifteen of these patients were explored, in 7 of whom the duodenum was not investigated because the surgeon was interested in some other abdominal condition. It is of interest that in the 8 patients in whom the condition was looked for, it was discovered in only 6. The writer believes that the condition is present more frequently than the x-ray reveals.

Bonar, T. G. D.: Congenital atresia of duodenum in child aged 13 months. Lancet **2**:822 (Oct. 12) 1935.
Nicholson, W. M.: Jaundice produced by a diverticulum of the duodenum. Bull. Johns Hopkins Hosp. **56**:305 (June) 1934.
Ockerblad, N. F., and Gonzales, N. G.: Duodenal fistula due to perinephritic abscess and following nephrectomy; case report and review of literature. Am. J. Surg. **28**:105 (April) 1935.
Lawson, J. D.: Duodenal diverticulosis. Am. J. Roentgenol. **34**:610 (Nov.) 1935.

LIVER

Boyce and McFetridge, from a clinical and experimental study, believe that so-called "liver death" is the result of the setting free of a water soluble toxin from the liver. This toxin particularly damages the kidney as well as other organs.

Ochsner and Debakey have made a study of liver abscess, especially amebic abscess. Reports already in the literature vary greatly on the incidence of amebic abscess in fatal cases of amebic dysentery. However, the average for 4,392 cases was 37.9 per cent. The abscesses were sterile in 83.9 per cent and amebas were found less often in the pus than in the walls of the lesions. Pathologic studies show that these abscesses more often involve the right lobe than the left. In their own cases fever was the most common symptom. Less frequently, the patient complained of chills, sweating, weakness, and so forth. The symptoms were intermittent in 7 per cent of the cases and remittent in 8 per cent. There was a previous history of diarrhea in 59.6 per cent. Local symptoms consisted of pain in the right upper quadrant, and usually enlargement of the liver. Jaundice was found to be rather infrequent. They state that the degree of leukocytosis is of value in distinguishing between amebic abscess and bacterial hepatitis or abscess. Usually, there is a moderate leukocytosis in amebic infection which may vary from 4,000 to 31,000, but averages 14,000. One expects to find a marked leukocytosis in bacterial infection of the liver. The x-ray may show a localized bulging of the diaphragm in the case of a subphrenic abscess. The mortality ranges from 4 per cent to 25 per cent, depending upon the type of treatment, and the writers advise conservative therapy whenever possible. Whenever an amebic abscess becomes secondarily infected, a fatal outcome is prone to follow. However, the best treatment is to give the patient a course of emetin, later aspirate the pus and follow with an intramuscular injection of emetin.

Chiray and Firmin have studied the acid-base equilibrium of the human bile which was obtained by biliary drainage and preserved under oil. As a result of these studies it appears that bile is a physicochemical system somewhat similar to blood. In general, zones of alkalosis and acidosis similar to those in the blood are indicated.

Boyce, F. F., and McFetridge, E. M.: So-called "liver death;" clinical and experimental study. Arch. Surg. **31**:105 (July) 1935.
Ochsner, A., and Debakey, M.: Liver abscess; amebic abscess; analysis of 73 cases. Am. J. Surg. **29**:173 (Aug.) 1935.
Chiray, M., and Firmin, P.: La réserve alcaline et l'acidité ionique de la bile humaine prélevée par tubage duodenal: note préliminaire. Arch. d. mal. de l'app. digestif. **25**:233 (March) 1935.

Anatomic Abnormalities

Boyden has made an interesting study of congenital anomalies of the gallbladder which may result in unusual appearances with cholecystography. There are two kinds of anomalies. One is what he calls the concealed or retroserosal type which is due to an early folding of the epithelial anlage of the gallbladder behind the peritoneal investment of the embryonic fossa vesicae felleae. The second is the serosal type which is due to aberrant folding of the fossa itself. These anomalies result in abnormal appearances of the gallbladder which may appear kinked, and if one is not familiar with them may suggest that the gallbladder is deformed by adhesions. Boyden found that 18 per cent of 165 individuals had marked kinking of the gallbladder. In 24 patients this occurred between the body and the infundibulum. In 6 it occurred between the body and the fundus. From physiologic studies, he does not believe that they are the cause of pain in the right upper quadrant.

Swartley and Weeder report an interesting abnormality in which there was a cyst of the common bile duct at the point at which the gallbladder empties.

McNamee reports 2 cases in which the gallbladder was in the liver tissue. In the first case the gallbladder was in the posterior portion of the right lobe of the liver, above the kidney. In the second case the gallbladder was entirely buried in liver tissue except for a small portion of the fundus. (That such abnormalities do occur is worth remembering, if the gallbladder is not visible at the time of operation.)

Boyden, E. A.: "Phrygian cap" in cholecystography; congenital anomaly of gallbladder. Am. J. Roentgenol. **33**:589 (May) 1935.
Swartley, W. B., and Weeder, S. D.: Choledochus cyst with double common bile duct. Ann. Surg. **101**:912 (March) 1935.
McNamee, E. P.: Intrahepatic gallbladder. Am. J. Roentgenol. **33**:603 (May) 1935.

Physiology of the Gallbladder

Halpert has studied the rate of resorption which occurs in the gallbladder. Methylene blue was given intravenously to rabbits, and the bile was forced to secrete against a constant hydrostatic pressure which he arbitrarily maintained at three different levels; namely, zero, 75 and 100 mm. above atmospheric pressure. The contents were collected from the common bile duct and removed at the end of the experiment. He found that there was a greater resorption of the fluid at higher pressures. He also found that the ratio of the rate of resorption to the volume of the gallbladder contents was the same under different conditions; namely, half the volume of the gallbladder contents were resorbed in one hour.

Hawkins and Whipple report the abnormalities which occur from exclusion of bile from the intestine. These observations have been made over a series of years on animals which Whipple has used for the study of the hematopoietic system. Exclusion of the bile from the intestine may cause:

1. Acute intestinal disturbances with rapid weight loss and even death.
2. Purpuric tendency with spontaneous bleeding.
3. General osteoporosis with multiple spontaneous fractures.
4. Cholelithiasis with obstruction in the fistulous tract.
5. Duodenal ulcer.

For tendency to diarrhea they advocate a diet rich in fat and protein, plus 50 cc. of fresh dog bile daily. Purpura usually develops three to four months after bile has been absent from the intestinal tract. For this condition they advocate 100 cc. of whole bile per day and it may take a week before improvement results. Osteoporosis is helped by the feeding of liver. Peptic ulcer occurred in 9 of 33 dogs in which the bile had been diverted from the intestine, and which lived more than three months.

Diseases of Gallbladder

Deissler and Higgins have shown that the extrahepatic biliary tract can become sensitive to anaphylaxis.

Gentile states that the hepatitis which occurs after cholecystogastrostomy depends upon the opportunity for infection which the biliary tract may acquire. Dogs' stomachs contain more bacteria than the average human stomach. He connected the gallbladder with the antrum and then separated the antrum from the body of the stomach. Then he connected the body with the jejunum. By this means the incidence of hepatitis was decreased. As a result of his studies, he is convinced that there is relatively little danger of hepatitis if the gallbladder is connected to the human stomach.

Stewart, Cantarow and Morgan have studied the renal changes which occur in biliary stasis.

They tied off the common duct of cats and later released the constriction. No extensive necrosis followed this operation, and there was no evidence of any consistent correlation between the renal changes and the stasis or decompression.

Halpert, B.; O'Connor, P. A., and Thompson, W. R.: Rates of resorption in the gall bladder. Am. J. Physiol. **112**:383 (June) 1935.
Hawkins, W. B., and Whipple, G. H.: Bile fistulas and related abnormalities; bleeding, osteoporosis, cholelithiasis and duodenal ulcers. J. Exper. Med. **62**:599 (Oct.) 1935.
Deissler, K., and Higgins, G. M.: Extrahepatic biliary tract during anaphylaxis. Am. J. Physiol. **112**:430 (July) 1935.
Gentile, A.: Cholecystogastrostomy and hepatitis; experimental study. Arch. Surg. **30**:449 (March) 1935.
Stewart, H. L.; Cantarow, A., and Morgan, D. R.: Renal changes in biliary stasis and decompression in cats. Arch. Path. **19**:807 (June) 1935.

Clinical Studies

Illingworth gives a good review of the subject of carcinoma of the gallbladder and reports a personal study of 30 cases. This is worth reading by anyone who is looking up this subject.

Illingworth, C. F. W.: Carcinoma of the gallbladder. Brit. J. Surg. **23**:4 (July) 1935.

PANCREAS

McNaught and Cox report the case of a 70 year old male, who died of lymphosarcoma, and in whom the pancreas surrounded the duodenum completely. Associated with this anomaly was a horseshoe kidney and an accessory spleen. They believe that this condition developed from the ventral pancreatic anlage. This is the forty-fourth reported case and this condition has been associated with other congenital anomalies in 25 per cent of the cases reported. It has not infrequently been the cause of duodenal obstruction.

Branch and Gross have reviewed the subject of aberrant pancreatic tissues occurring in the gastrointestinal tract, and add 24 cases to approximately 200 already in the literature. The pancreatic tissue was distributed as follows: In 3 instances the tissue was found in the stomach; in 10 it was located in the duodenum; in 4 in the jejunum; 1 in the ileum, and in 6 it was present in a Meckel's diverticulum. One caused pyloric obstruction, and in 3 cases it was the site of ulceration in the stomach and duodenum.

Kaplan and Chaikoff have made a further study of the liver lipids in depancreatized dogs which have been maintained with insulin. They confirm the observation of other investigators that abnormal amounts of lipids occur in the liver of such animals and that the fats disappear from the blood abnormally rapidly.

De Tarnowsky and Sarma discuss the surgical treatment of chronic pancreatitis on the basis of 30 cases. The following symptoms were present: dyspepsia in 95 per cent, pain in the right upper quadrant in 55 per cent, nausea and vomiting in 43 per cent, weakness and anorexia in 24 per cent. Bile was present in the urine in 18 per cent. Slight icterus was seen in 18 per cent, severe icterus in 12 per cent.

Ransom gives his findings in 16 patients suffering from carcinoma of the body and tail of the pancreas. The most common symptoms were severe pain which frequently occurred in crises, and rapid emaciation without jaundice. A tumor was palpated in half the cases. Laboratory studies were of very little help, and the roentgen ray gave a clue to the condition in only one-fourth of the cases. The condition was mistaken for carcinoma of the colon more frequently than for anything else. The average duration of life for these patients was 4.7 months after being seen, and 10.2 months from the beginning of the symptoms. Surgery afforded relief frequently, but symptoms usually recurred before death.

McNaught, J. B., and Cox, A. J., Jr.: Annular pancreas: report of a case with a simple method for visualizing the duct system. Am. J. Path. **11**:179 (Jan.) 1935.
Branch, C. D., and Gross, R. E.: Aberrant pancreatic tissue in the gastro-intestinal tract; report of 24 cases. Arch. Surg. **31**:200 (Aug.) 1935.
Kaplan, A., and Chaikoff, I. L.: Liver lipids in completely depancreatized dogs maintained with insulin. J. Biol. Chem. **108**:201 (Jan.) 1935.
de Tarnowsky, G., and Sarma, P. J.: The surgical treatment of chronic pancreatitis. Ann. Surg. **101**:1342 (June) 1935.
Ransom, H. K.: Carcinoma of the body and tail of the pancreas. Arch. Surg. **30**:584 (April) 1935.

JEJUNUM AND ILEUM

Nasset and Pierce have studied the influence of peptones on the secretion of the succus entericus. They found that Witte's peptone contains a potent secretagogue which, given by mouth or vein, acts on the glands of the jejunum. This substance can be extracted with 70 per cent ethyl alcohol. It is digested by erepsin or trypsin, and is thermostable. It seems to be present only in Witte's peptones, as five other peptones which were studied showed little secretagogic activity.

Mackie, Miller and Rhoads believe that the small intestine shows characteristic changes by the x-ray in patients suffering from sprue. The mucosal folds of the duodenum appear thickened and the lumen is irregularly dilated. The valvulae conniventes of the jejunum are also thickened, and are more widely separated than normal. The barium passes through the jejunum slowly and irregularly. Pockets of barium can be seen in which there is no evidence of peristalsis.

Blackman reports the findings at necropsy in a 12 year old Negress who died of acute enteritis of two or three days' duration, after a large Thanksgiving dinner. Autopsy showed marked lesions in the upper part of the jejunum which decreased in severity and frequency as one approached the ileocecal valve. The upper intestine was lined with a membranous cast. The whole area was infected with *Staphylococcus al-*

bus. Microscopic examination revealed a diffuse superficial necrosis with acute inflammation of the entire mucosa of the jejunum and ileum. Septicemia was the cause of death.

Price followed a patient with a carcinoid tumor of a Meckel's diverticulum. He was able to find, in the literature, only two reports of a similar condition. The patient died of an intestinal obstruction at the age of 54 years.

Nasset, E. S., and Pierce, H. B.: On the influence of peptones and certain extracts of small intestine upon the secretion of succus entericus. Am. J. Physiol. **113**:568 (Nov.) 1935.
Mackie, T. T.; Miller, D. K., and Rhoads, C. P.: Sprue—roentgenologic changes in the small intestine. Am. J. Trop. Med. **15**:571 (Sept.) 1935.
Blackman, S. S., Jr.: Acute staphylococcal infection of the jejunum and ileum. Bull. Johns Hopkins Hosp. **57**:289 (Nov.) 1935.
Price, I.: Carcinoid tumor of Meckel's diverticulum: report of case. Brit. J. Surg. **23**:30 (July) 1935.

COLON

Sparks and Collins have repeated the work of Hargreaves, Fletcher, and Dickson when studying the effect of vitamin B on the tonus of the colon. The degree of tonus was determined by measuring the amount of barium needed to fill the colon as seen under the fluoroscope. The normal volume of the colon of adult rats is from 3.0 to 4.5 cc. The volume of the colon of rats maintained on a diet deficient in vitamin B_1 increased strikingly in 70 per cent of the animals. No such change occurred in a control group maintained on a complete synthetic diet.

Opper reports the case of a 22 year old boy who died from a congenital megacolon. The intestine was of enormous size and the history and autopsy point to compression of the heart and lungs as the cause of death.

Stabins, Morton, and Scott believe that spinal anesthesia is of use in the diagnosis and treatment of megacolon and obstinate constipation. By using spinal anesthesia in patients in whom the cause of a large colon or obstinate constipation is not clear, one is able to demonstrate when an excess of sympathetic activity plays a dominant rôle.

Mitchell has made a study of the innervation of the distal colon by dissection on 15 stillborn babies. Included in this article are 6 photographs and 2 sketches. He concludes that the distal colon receives its nerve supply from the inferior mesenteric plexus and the hypogastric plexus. The former, or inferior mesenteric plexus supplies mainly, or possibly, all the sympathetic fibers. The hypogastric nerves and plexus supplies both sympathetic and parasympathetic fibers, but chiefly the latter.

Slavin calls attention to the fact that paralyzing the diaphragm by phrenicotomy may result in the liver receding from the diaphragm. In one of two cases which he has observed, the colon assumed a position between the liver and the diaphragm. Although the patient had no symptoms, colonic gas was noticed in this area.

Several articles have been written on the intestinal flora.

Petran from a study on monkeys and dogs, was unable to demonstrate that bile had any effect on the intestinal flora. On the contrary, lactose stimulated the growth of acidophilus organisms.

Jordan and Burrows have made the rather interesting observation that many of the organisms found in the intestinal tract can produce an enterotoxin when grown under suitable conditions. This is true of the staphylococcus, streptococcus, proteus, and colon bacteria, the aerogenous organisms and Salmonella aertrycke. Organisms which have been cultured for some time and show no evidence of this tendency, can regain it if they are cultured in a suitable way. (Such a discovery suggests that it is possible for persons to develop a condition of auto-intoxication, but further work must be done before one can improve the treatment of such a condition when it is suspected.)

Althausen et al. have studied the flora in patients with obvious intolerance for carbohydrates. They have been unable to show any change from the normal flora. (This finding would agree with the observations of Schmidt and Strassburger, that carbohydrate intolerance depends upon a digestive disturbance in the upper intestine.)

Except in isolated instances, little attention has been paid to the fungi as a possible cause of digestive disturbances. Therefore, it is of interest that Reed and Johnstone have attempted to investigate this problem. They have studied the fungi present in the intestines of patients who were in otherwise good health and were suffering from various intestinal disturbances. They were able to isolate fungi from the intestines of about 52 per cent of the individuals they studied. However, the authors were unable to show that the presence of these fungi was the cause of, or influenced, any disease process that might be present.

Rainey and Cole conclude that the rectal strictures which are so commonly found among Negro women are the result of lymphogranuloma inguinale.

Sparks, M. I., and Collins, E. N.: The rôle of vitamin B1 in tonus of the large intestine. Am. J. Digest. Dis. & Nutrition **2**:618 (Dec.) 1935.
Opper, L.: Congenital megacolon. Am. J. Path. **11**:365 (March) 1935.
Stabins, S. J.; Morton, J. J., and Scott, W. J. M.: Spinal anesthesia in the treatment of megacolon and obstinate constipation. Am. J. Surg. **27**:107 (Jan.) 1935.
Mitchell, G. A. G.: The innervation of the distal colon. Edinburgh M. J. **42**:11 (Jan.) 1935.
Slavin, P.: Interposition of the colon following induced phrenic paralysis. Am. J. Roentgenol. **33**:481 (April) 1935.
Petran, E.: Intestinal flora of monkeys and dogs during digestion and following direct introduction of food substances into the cecum, and into isolated segments of the bowel. J. Infect. Dis. **57**:296 (Nov.-Dec.) 1935.
Jordan, E. O., and Burrows, W.: The production of enterotoxic substance by bacteria. J. Infect. Dis. **57**:121 (July-Aug.) 1935.
Althausen, T. L.; Gunnison, J. B.; Marshall, M. S., and Shipman, S. J.: Carbohydrate intolerance and intestinal flora; clinical study based on 60 cases. Tr. Am. Gastro-Enterol. A. **36**:143, 1933. Arch. Int. Med. **56**:1263 (Dec.) 1935.

Reed, A. C., and Johnstone, H. G.: A clinical study of intestinal fungi. Am. J. Trop. Med. **15**:155 (March) 1935.
Rainey, W., and Cole, W. H.: Lymphogranuloma inguinale; its relation to stricture of rectum. Arch. Surg. **30**:820 (May) 1935.

DYSENTERY AND DIARRHEA

G. S. de Paula e Silva found 22 instances of intestinal infestation with Giardia in 572 patients observed in Brazil, an incidence of 3.8 per cent. The diagnosis was made in each case by drainage of the duodenum.

He points out that one does not always find evidences of the parasite in the stools when they are present in the duodenum. He lists the symptoms as follows:

1. Severe pain similar to biliary colic (but he was not able to rule out cholelithiasis in most of these cases).
2. General dyspeptic symptoms.
3. Of 22 patients who complained of no definite symptoms, 9 had regular bowel movements, 4 had alternating constipation and diarrhea, 6 had attacks of diarrhea and 2 had obstinate constipation.
4. Systemic symptoms which could not be associated with the intestinal tract. (Apparently it is difficult to determine how important this parasite is in the production of gastrointestinal symptoms. One can only say that at times it is responsible for symptoms, and the possibility of its presence should be kept in mind.)

MacPhee and Walker discuss the general problem of intestinal infestation with *Giardia intestinalis*. The authors have found only 6 cases of this condition in 732 patients upon whom a routine bile drainage was done, an incidence of .82 per cent. This is lower than a previous report by Burke who studied children in the Childrens' Hospital in Boston. This author found an incidence of 14 carriers among 80 miscellaneous medical admissions. The 6 cases reported by the present authors complained of abdominal symptoms of various types, so that no characteristic symptoms could be associated with the infection.

It must be accepted that amebic dysentery has become endemic in the United States. Hence, the study of Wenrich et al. is of interest, on the incidence of *Endameba histolytica* among a group of college freshmen. Thirty-four and five tenths per cent of 1,060 freshmen were found to harbor one or more kinds of protozoa and 4.1 per cent were shown to have *Endameba histolytica*. In spite of this fact, no cases of amebic dysentery were reported in the university among the carriers or noncarriers.

Callender stresses the type of stool that the patient with amebic dysentery is having as a guide to the medicine which should be used in its treatment. He believes that the arsenicals increase the amount of inflammatory exudate in the stools and suggests that the presence of pus is evidence of a secondary infection. When a patient with amebic dysentery is showing much pus, emetin is indicated, whereas, the arsenicals such as carbarsone are of value when little pus is present.

From a review of 100 cases, Bockus discusses the pathogenesis of idiopathic ulcerative colitis. He classifies the disease into:

1. The chronic relapsing type.
2. The chronic continuous type.
3. The acute fulminating type.
4. The early healing type.

He believes that a positive diagnosis can be made only by the use of the sigmoidoscope, but declares that a barium enema is of paramount importance in ascertaining the extent and degree of involvement. In a discussion of the etiology, he states that the habitus of the individual seems of little consequence. He stresses the necessity of giving consideration to the emotional or psychogenic factor in patients with this disease. From a study of foci of infection he has not been able to determine any type of infection which is common to the group. Animal inoculations in his clinic have failed to establish the specificity of the streptococcus in ulcerative colitis. He favors the contention of Hurst and others, that dysentery bacilli play an important rôle in the initiation of chronic ulcerative colitis, and states that these organisms must be considered as possible initial invaders.

Bargen and Dixon believe that when carcinoma develops on the site of chronic ulcerative colitis it is best to resect the entire colon rather than to attempt a local resection.

Haskell and Cantarow report further results on the treatment of ulcerative colitis with calcium supplemented by parathyroid therapy. They administer 60 grains of calcium gluconate 3 to 4 times daily, 3½ to 4 hours after meals. Parathormone is injected intramuscularly, the average adult dose being 20 units. These injections are repeated at intervals of 48 to 72 hours, depending upon the severity of the symptoms. These patients have been observed for periods of from 2 to 6 years. Five have remained essentially symptom-free. Brief remissions occurred in two instances, which however, promptly responded to the same form of treatment. Sixteen patients have been observed from 6 months to 4 years. Eight became clinically well, 7 were relieved of their severe symptoms, and 1 patient was not benefited. Although the response to treatment is usually prompt, definite improvement occurred in 3

cases, only after 3 months of continuous calcium and parathyroid administration.

In the April and May issues of the *American Journal of Digestive Diseases and Nutrition,* is a comprehensive article by Sibrand Lups, of Groningen, Holland, which has been translated and edited by A. J. Baker, on the subject of Vaccine Therapy in Ulcerative Colitis. This article is too long to summarize thoroughly.

Denison and deHoll believe that the acute diarrheas of infancy should primarily be divided into gastrointestinal infections due to dysentery bacilli and disturbances of function from numerous causes. The authors have isolated 157 cultures suggestively characteristic of dysentery bacilli. In a study of 35 patients with infectious diarrhea, dysentery bacilli were recovered from 63 per cent of the stools from which cultures were taken during the first five days of illness. After the fifth day, the chances for recovering the organism rapidly diminished although the majority of the stools continued to show blood and pus for fifteen days longer.

Meleney and Harwood report an unusual case of infection with the larvae of the soldier fly. So far as these authors were able to determine, this is the second case of its kind reported. The patient had interesting symptoms which included three fainting spells, and a feeling of something biting him or eating him, first in the region of the stomach and later in the rectum. Later the larvae were passed and it was then that the diagnosis was made.

G. S. de Paula e Silva: A clinical review of giardiasis; 22 cases observed during study of 572 private patients. Am. J. Digest. Dis. & Nutrition **2**:350 (Aug.) 1935.

MacPhee, L., and Walker, B. S.: Intestinal giardiasis in New England, with notes on its pathogenicity and symptomatology. Am. J. Digest. Dis. & Nutrition **1**:768 (Jan.) 1935.

Wenrich, D. H.; Stabler, R. M., and Arnett, J. H.: Endamoeba histolytica and other intestinal protozoa in 1,060 college freshmen. Am. J. Trop. Med. **15**:331 (May) 1935.

Callender, G. R.: Amebic dysentery; exudate as a guide to treatment. Am. J. Trop. Med. **15**:189 (March) 1935.

Bockus, H. L.: The pathogenesis of idiopathic ulcerative colitis. Delaware State M. J. **8**:1 (Jan.) 1936.

Bargen, J. A., and Dixon, C. F.: Chronic ulcerative colitis with associated carcinoma; progress in management. Arch. Surg. **30**:854 (May) 1935.

Haskell, B., and Cantarow, A.: Further studies in calcium and parathyroid therapy in chronic ulcerative colitis. Am. J. M. Sc. **190**:676 (Nov.) 1935.

Lups, Sibrand: Vaccine therapy in ulcerative colitis. Am. J. Digest. Dis. & Nutrition **2**:65 (April) 1935; **2**:139 (May) 1935.

Denison, G. A., and deHoll, G.: Bacillary dysentery in infants and children; clinical and bacteriologic study of 35 cases. J. Infect. Dis. **56**:124 (March-April) 1935.

Meleney, H. E., and Harwood, P. D.: Human intestinal myiasis due to larvae of soldier fly, Hermetia illucesis, Linné (Diptera, Stratiomyidae). Am. J. Trop. Med. **15**:45 (Jan.) 1935.

APPENDIX

Snyder et al. have made an interesting study of the association of pylephlebitis and appendicitis. They summarize this work as follows: "Chills in appendicitis cases operated upon early (within thirty-six hours of onset) do not necessarily signify the development of pylephlebitis. Two and one tenth per cent of all our cases of acute appendicitis had chills. Acute appendicitis of several days' duration associated with chills is likely to be followed by pylephlebitis. This occurred in forty per cent of such patients in our series. The incidence of pylephlebitis following appendicitis in our hospital was .3 of one per cent. Approximately one patient in six having chills during the attack of appendicitis developed pylephlebitis. When pylephlebitis occurs it is usually in neglected cases or in those in which the appendix is not removed early. The average time after the onset of symptoms of appendicitis before operation was performed was 7.7 days. The average time in our series after the onset of symptoms of appendicitis before pylephlebitis developed to a point where the diagnosis could be made, was 13.3 days. Histological study of the appendix mesentery is of little value in determining the development of pylephlebitis. The clinical evidence and the gross appearance of the local lesion at the time of operation are often characteristic. Ligation of the ileocolic vein is recommended in carefully selected cases."

Mead has discussed mesenteric lymphadenitis simulating acute appendicitis. He studied the size of the glands in these cases without obtaining results of any practical value. However, he includes a good bibliography which will be of value to anyone investigating this condition.

Snyder, W. H.; Hall, M. G., and Allen, A. W.: The association of pylephlebitis and appendicitis. New Eng. J. Med. **212**:183 (Jan. 31) 1935.

Mead, C. H.: Mesenteric lymphadenitis simulating acute appendicitis; quantitative study of size of normal mesenteric lymph nodes. Arch. Surg. **30**:492 (March) 1935.

A DEPARTMENT OF FORENSIC MEDICINE

The Department of Forensic Medicine of the New York University College of Medicine has been reorganized under the direction of Dr. Harrison Martland, who succeeded Dr. Charles Norris as Professor of Forensic Medicine in January, 1936. In addition to undergraduate work, the department has developed graduate instruction leading to the degree of Med.Sc.D, and short, intensive courses in specialized branches of medico-legal work. The Charles Norris Fellowship in Forensic Medicine has been established and is open to candidates applying for work toward the degree.

WILL WEYMOUTH HAVE A HOSPITAL?

By the will of Laban Pratt, a $300,000 fund was left to the town of Weymouth, about ten years ago, for the purpose of providing a hospital. This fund has grown to $461,000, according to published statements, but will revert to the heirs of the donor if not utilized by the town.

The Attorney General has issued a command to the selectmen of Weymouth to show cause why no constructive action with respect to this bequest has been taken. A committee will be appointed to consider the local conditions and report at a town meeting.

The situation seems to be almost unique, for most communities would take advantage of a gift of this character.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22421

PRESENTATION OF CASE

First Admission. A 73 year old postman was admitted complaining of pain in the abdomen.

The patient was perfectly well until 3 days before coming to the hospital, at which time he lifted a heavy weight and noted shortly afterward a sensation of soreness localized to the right upper quadrant. On the first day of his illness he vomited large amounts of yellowish bitter fluid. There was persistent nausea and marked anorexia. Bowel movements, which usually occurred daily, had not occurred for 2 days before entry. There were no other associated symptoms.

Physical examination showed a slender, well-nourished, elderly man in no apparent discomfort. There was moderate sclerosis of the peripheral vessels. The lungs were clear and the heart normal. The blood pressure was 160/80. The abdomen was distended and tympanitic. There was marked tenderness and spasm in the right upper quadrant and a questionable mass was palpated in this region.

The temperature was 99.8°, the pulse 130. The respirations were 22.

Examination of the urine showed a specific gravity of 1.030 with a slight trace of albumin. The sediment was negative. The blood showed a white cell count of 23,000. Examination of the stools was negative.

A plain film of the gallbladder region showed no shadow significant of disease of that organ.

Subsequently the patient's temperature showed a daily rise of 100° and his pulse and temperature remained elevated. Subjectively, however, he became more comfortable and on the seventh day a laparotomy was performed. At operation a large pericholecystic abscess containing several ounces of odorless pus was found and the gallbladder wall appeared to be necrotic. The gallbladder was opened and a large amount of detritus and a dozen small stones were removed. Normal yellow bile was aspirated from the cystic duct. A tube was sutured into the gallbladder and drains were placed in the abscess cavity. The patient had a stormy immediate postoperative course during which he had collapse of the right lower lobe. He made a good recovery, however, and was discharged on the twenty-seventh hospital day.

Second Admission, one year later.

The patient remained quite well after leaving the hospital until one week before re-entry. At this time he began to notice some gaseous eructation, indigestion, and a gradually increasing jaundice. There was no abdominal pain but he did have some aching pain in the lower back. There were no chills, fever, nausea, or emesis. The upper portion of his abdomen appeared to be hard and bulging. There was some increasing readiness of fatigue and a loss of about 10 pounds during the preceding 2 months. Bowel habits and micturition were unchanged.

Physical examination showed a deeply jaundiced man in no acute discomfort. The heart and lungs were unchanged. The abdomen was distended, tense, and tympanitic. The scar of the previous operation was well healed. Motion of the spine caused pain in the lower portion of the back. Rectal examination showed a rather firm, medium-sized, nonfixed prostate, the left lobe of which was slightly larger than the right.

The temperature was 100°, the pulse 110. The respirations were 20.

Examination of the urine showed a trace of albumin but was otherwise negative. The blood showed a red cell count of 2,990,000, with a hemoglobin of 60 per cent. The white cell count was 8,700, 61 per cent polymorphonuclears. The smear showed marked poikilocytosis, anisocytosis, and a rare stippled red blood cell. A reticulocyte count was 6.9 per cent. Hematocrit studies showed the average red cell volume to be considerably below normal. Stool examinations showed an occasional positive reaction to the guaiac test but were otherwise negative. Bile was present. A van den Bergh test showed 11 milligrams bilirubin. Duodenal drainage sediment contained moderate amounts of bilirubin crystals and there was a very high pigment content.

X-ray examination showed numerous rounded areas of new bone formation in the bones of the pelvis, lumbar spine, and thorax. The large bowel was filled with gas down to the rectum. A chest film was negative.

The patient continued to run a low-grade febrile course. He was given liver extract and the reticulocyte count rose to 9.0 per cent and the red cell count of the blood rapidly dropped to 1,680,000, with a hemoglobin of 45 per cent. The white cell count rose to 18,000, with 52 per cent polymorphonuclears, 4 per cent myelocytes, 29 per cent lymphocytes, 2 per cent monocytes, and 13 per cent normoblasts. Genito-urinary consultants did not believe that the prostate was malignant. The icterus index gradually dimin-

ished to 4.6 milligrams and the patient was discharged on the eleventh hospital day.

Final Admission, twelve days later.

Following his discharge the patient rapidly lost weight and strength. The jaundice diminished rapidly and was replaced by a pronounced pallor. The pain in the lower back increased and became more troublesome.

Physical examination showed an obviously ill man with faint yellowish pallor. A few small hemorrhagic spots were noted in the conjunctival sacs and buccal mucous membrane. There were no other significant changes. The blood pressure was 135/60.

The temperature was 101°, the pulse 120. The respirations were 22.

Examination of the urine was negative. The blood showed a red cell count of 1,300,000, with a hemoglobin of 45 per cent. The white cell count was 22,000, 80 per cent neutrophils. There were a few immature myeloid cells and normoblasts.

The temperature rose gradually to 103° and the pulse to 160. He became rapidly weaker and died on the fourth hospital day, a little more than a year after his first entry.

Differential Diagnosis

Dr. Wyman Richardson: The first question we have to answer is whether trauma had anything to do with this patient's entry.

Aside from a diaphragmatic hernia as the result of trauma, which I think would be very rare, I do not see the relationship between his lifting a heavy weight and the symptoms. There is some suggestion of intestinal obstruction.

"On the first day of his illness he vomited large amounts of yellowish bitter fluid." If this is accurate it certainly would rule out pyloric obstruction because "yellow bitter fluid" presumably would contain bile.

"The blood pressure was 160/80." He has a high pulse pressure as so many of the people with peripheral arteriosclerosis have.

As you know, palpation of the upper quadrants is extremely difficult. It is always difficult to be sure of spasm in the right upper quadrant but in this case the note is made that there was marked tenderness and spasm in the right upper quadrant. We will take it as a fact that there was some peritoneal irritation in that region. I think we will have to let the questionable mass go. If there was spasm it would be even more difficult to feel the mass. So we have to try to explain what may be intestinal obstruction and what may be peritoneal inflammation in the right upper quadrant.

The physical examination is really lacking in one regard; that is, no rectal examination was done. At least there is no record in the summary. We will assume that it was done and nothing definite was discovered. We would like to know whether there were feces in the rectum or marked rectal tenderness.

"Examination of the stools was negative." That is important. He must have passed stools in the hospital. We do not know whether he passed gas but that is definite evidence that he did pass stools.

I find it difficult to be dogmatic about the diagnosis.

The supposition is that he has something in the right upper quadrant. I suppose it might be an ulcer. There is no suggestion of ulcer history and no suggestion of perforation with peritonitis, unless it is well localized. Usually with peritonitis there is an extension of the signs and symptoms to the lower abdomen. From that story one would be inclined not to wait long for operation. I am not at this point able to guess what was done. We will read on and see what the subsequent course was.

It says here that the gallbladder wall appeared to be necrotic. That leaves us with the distinct suspicion that possibly some large stone escaped through the gallbladder wall; but there is no mention made of any large stone being found. We will look with interest at the future course of this patient to see if the large stone is lost somewhere in the abdomen.

A Physician: Why do you think it was ruptured?

Dr. Richardson: It may have been intact.

Dr. Mallory: I do not think they found any gross communication between the pericholecystic abscess and the gallbladder. The gallbladder was reported as containing normal looking bile as well as gallstones and the pericholecystic abscess contained pus which was not bile stained.

A Physician: It could not, therefore, have been a communication between the gallbladder and the abscess cavity.

Dr. Richardson: That is a rather curious situation, it seems to me, to have a definite abscess around a gallbladder which showed so little evidence of being intrinsically infected. Perhaps the gallbladder is not the cause of the abscess at all. We have to consider other causes of that abscess. He had no gastrointestinal x-ray during his stay in the hospital. Just where the pus in that region came from, I do not feel sure, unless from the gallbladder. He still might have had a ruptured localized ulcer. There is no evidence of pylephlebitis that I can see.

With regard to the second admission, it would be perfectly all right to have a gradually increasing jaundice, but all the important features in regard to the symptoms, or many of them, do not appear. What we want to know particularly is whether this is obstructive jaun-

dice or not. We may be able to tell from the laboratory findings later. One would like to know about that and whether the patient noticed bile in the urine. One cannot be sure whether it is intrinsic liver disease or obstructive jaundice from some process causing fibrous obstruction as a result of the abscess or operation or some other process.

The abdomen was distended but the note that it was tympanitic would seem to rule out abdominal fluid as the cause of the distention. There was no suggestion of obstruction from the history.

"Motion in the spine caused pain in the lower back." That may or may not mean something. A man with malignant metastases of the spine generally complains of not being able to turn over in bed. That is one of the major complaints. I still have no good suggestion as to what this process is. I was thinking of the possibility of bile peritonitis. There is no definite evidence of fluid in the abdomen.

The blood examination shows an anemia with variation in the size of the cells, with more of the cells being smaller than normal, an anemia associated with an active bone marrow as shown by nearly 7 per cent reticulocytes. It is a bone marrow putting out young cells and at the same time putting out abnormal cells as shown by variation in shape and size. A "rare stippled cell" I consider simply evidence of an active bone marrow. Since it is a "rare" stipple cell it does not suggest necessarily lead poisoning or other disease.

If we just took the blood examination without any thought of the history or patient or anything else, it would be consistent with hemolytic jaundice of some type. In regard to the urine, it says it was negative except for the albumin. Presumably there was no bile as such in the urine, although probably there may have been urobilinogen. I do not believe they tested for it. That is not the anemia of infection, and we rule out obstructive jaundice. Incidentally the physical examination did not reveal any definite mass, nor did it tell us what the size of the liver was.

The x-ray man apparently considered the bones to be characteristic of metastatic malignancy, particularly of the type that is seen with carcinoma of the prostate, since it was an osteoplastic rather than an osteoclastic lesion.

I was wondering whether the anemia could be called the myelophthisic type with replacement of marrow by tumor tissue. You often get a blood picture showing very active blood formation in this condition. This is not pernicious anemia in relapse because there is too much red cell activity for the degree of anemia.

We have to stick to what we have that is definite, and the definite things are pericholecystic abscess, an interval of a year with the appearance of jaundice, an anemia which shows considerable red cell activity and an x-ray examination suggesting metastatic disease of the bone with some discussion in regard to the prostate. The anemia could be due to metastatic disease alone. I do not believe it was the so-called primary type. It certainly was not pernicious anemia. It seems very unlikely that this man would develop hemolytic jaundice. I do not see any point in bringing in that diagnosis. We must assume he has intrinsic liver pathology with considerable destruction of liver tissue that may result in anemia, and there is probably involvement of the bone marrow itself by some malignant process. He may have died of liver failure although the jaundice all cleared up. There is one other possibility here, that is drugs. No mention is made in the history, so I would rule that out.

I really have no diagnosis to offer in this case. In the first place I do not feel sure as to the cause of his abscess. He could have a primary malignancy of the liver which may occur in these cases that have had gallstones of long standing. I am going to say that he had malignant disease with liver destruction, with the source of the malignancy not definite, though I am going to express the suspicion that we may be dealing with primary malignancy of the liver.

A PHYSICIAN: Is it not possible that the first process could be an independent affair and separate from the malignancy?

DR. RICHARDSON: I think it is possible with a year of relative freedom in between; but it is all such an obscure story that one feels a proper diagnosis had better include that episode as a part.

A PHYSICIAN: Why should the jaundice clear up rapidly twice if not an obstructive one?

DR. RICHARDSON: I do not know; jaundice often waxes and wanes in conditions that are not obstructive.

A PHYSICIAN: If it were an obstructive jaundice in the face of some of the things we have been thinking of, I would expect it not to clear up but rather to become more intense and persistent. From the evidence that we have, which is scanty, it appears not to be an obstructive jaundice.

CLINICAL DIAGNOSIS

Metastatic carcinoma of the prostate.

DR. WYMAN RICHARDSON'S DIAGNOSIS

Malignant disease (? primary in the liver).

ANATOMIC DIAGNOSES

Carcinoma of the prostate with metastases to the vertebrae, the liver, and the regional and infrahepatic lymph nodes.

Benign hypertrophy of the prostate, median bar.
Lipomatous polyp of the ascending colon.
Pleuritis, chronic fibrous, left.
Peritonitis, chronic fibrous, localized.
Chronic cholecystitis.
Cholelithiasis.
Diverticulosis of the colon.
Operative scar, cholecystostomy.

Pathologic Discussion

Dr. Tracy B. Mallory: I might mention the fact that this patient was a doctor's father and almost anyone in medicine knows that fathers of doctors always have the most difficult conditions to make head or tail of. The men on the ward finally decided to side with the x-ray department against the genito-urinary department and, in spite of the statement of the head of the genito-urinary service that he did not think the prostate was malignant, they finally signed the patient out with the diagnosis of carcinoma of the prostate with metastases, and that was what we found at autopsy. I would be embarrassed if you were to ask me to demonstrate that fact, however. The prostate showed an ordinary benign hypertrophy except for one quite small area not more than 5 millimeters in diameter that was located deep in the gland, so it was natural that the examining finger in the rectum should have felt the benign hypertrophy and not this single firm nodule. We made a frozen section of it at the time of the autopsy and several of us who saw it agreed there was no doubt that it was malignant. We put through ten other blocks, however, for paraffin sections and could not find cancer in any of them. However, I feel quite certain that the cancer was primary there. Tumor was very obviously present throughout the bone marrow and was so characteristic in type that it would be fairly safe to commit oneself to a diagnosis of prostatic malignancy on the sections of the vertebrae alone.

The findings as regards the liver and bile passages were a little disappointing. They had left his gallbladder in at the previous operation and there were of course adhesions around it. The bile ducts were all patent. The gallbladder contained bile and inside the gallbladder and within all the small bile ducts in the liver were very numerous crystals of bilirubin, essentially similar to the ones that had been aspirated from the duodenum at one time, but nothing else. The liver parenchyma was normal, so that the only explanation I can give for his jaundice is that this crystalline bilirubin was thick enough in some places to make a sort of mud in the bile ducts. I believe I have seen such a phenomenon produce jaundice in other cases and perhaps variations in the concentration of the bile from time to time permitted occasional washing out of the mud. That is the type of thing we see very often, as Dr. Richardson has brought out, in hemolytic jaundice. I find it hard to understand, however, why this man should have had any excessive hemolysis. He appeared to represent apparently an obvious case of anemia from displacement of very large amounts of bone marrow by tumor. That the amount displaced must have been extremely large could be inferred from one unusual finding. The liver, I have said, was normal, but it was normal only in so far as any evidence of degeneration or necrosis was concerned; it was unusual in that it had a great many foci of hematopoiesis in it made up of erythroblasts and normoblasts. The same thing was found in the spleen. With severe myelophthisic anemia, after the bone marrow is apparently unable to provide enough blood cells, one always may get a reversion to the fetal type of blood formation and liver and spleen again begin to form blood cells. You frequently see it in children and occasionally see it in young adults. It is rare, however, in middle age onward and I am not sure I can remember another patient of seventy in whom the liver and spleen were forming blood. It is certainly unusual and the stimulus to blood cell formation must have been terrific to bring that to pass. The involvement of his vertebral column at the autopsy was so extensive that we found practically no areas of blood formation in any one of several vertebrae that were examined. The marrow was completely replaced by tumor.

A Physician: Is there any explanation of the abscess?

Dr. Mallory: No; that was completely healed at the time of autopsy.

A Physician: Do you think he had tumor invasion of the bone marrow at the time of his first entry?

Dr. Mallory: One can only guess. Cancers of the prostate on occasion can grow very fast but by and large they are among the slowest growing types of malignancy.

Have you any suggestions, Dr. Richardson?

Dr. Richardson: No, the hemolytic element here seems extraordinary. It is interesting and unquestionably I think we have to assume he had excessive hemolysis.

I wonder if it is possible for a man to live seventy years with a latent hemolytic jaundice that is brought out by other disease.

Dr. Mallory: We have learned that the spleens of hemolytic anemia cases are very characteristic histologically and there was none of the characteristic picture in this case.

I forgot to mention one thing which might easily have complicated the story much more for Dr. Richardson. He had a lipoma of the large bowel and numerous diverticula of the

large bowel. If they had done a barium enema and shown up the bowel lesion we would have had a still better case.

CASE 22422

Presentation of Case

A 66 year old English housewife entered complaining of abdominal pain.

About one year before entry the patient first noted that her stools appeared to be grooved. There was constipation of many years' duration. The condition continued without much change and about 3 months ago she began to have attacks of cramp-like abdominal pain, each attack of which lasted for about 3 days. The pain was most marked in the right upper quadrant but was not associated with nausea or vomiting. There was no weight loss. Occasionally the stools appeared to be blood-streaked.

The patient lived in India during her childhood and had had cholera, smallpox, and "jungle fever". Twenty-two years ago she was operated upon for a fibroid tumor and her menses ceased abruptly after this operation.

Physical examination showed a well-developed and nourished, obese, elderly woman, weighing 240 pounds, in no evident discomfort. There was kyphosis of the dorsal spine with slight chest deformity. The heart was not enlarged but a soft apical systolic murmur was audible. The blood pressure was 198/50. The lungs were clear. Abdominal examination showed a well-healed lower midline scar but the abdominal wall was soft, flaccid, and there were no palpable masses or tenderness. A vaginal examination was essentially negative. Rectal examination showed a few small, nontender hemorrhoids and proctoscopy exhibited a normal rectal mucosa up to the lower portion of the sigmoid. Beyond this point it was impossible to pass the proctoscope but no abnormality of the bowel could be recognized except for considerable narrowing and a plug of mucus occluding the passage.

The temperature, pulse, and respirations were normal.

Examination of an uncatheterized urine specimen showed a specific gravity of 1.020 with a trace of albumin. The sediment contained occasional red and white blood cells but was otherwise negative. The blood showed a red cell count of 4,900,000, with a hemoglobin of 85 per cent. The white blood count was 7,600, 61 per cent polymorphonuclears.

A chest x-ray showed the diaphragm in the usual position. There was relative brightness of the lung fields but the pulmonary vascular markings were increased, and the left ventricle was prominent. The aortic knob was sclerotic and the aorta tortuous.

On the third hospital day a laparotomy was performed.

Notes on the History

Dr. Robert R. Linton: In reviewing this case it seems to me the most obvious thing that was troubling this patient was some degree of intestinal obstruction. The history is not a very adequate one. This makes it difficult to make a diagnosis.

The first statement I should like to comment upon is that she had noticed that her stools were grooved. I doubt if that means anything at all. It used to be taught that ribbon-like stools were diagnostic of carcinoma of the rectum, but I do not believe that the type of the stool has anything to do with carcinoma of the rectum or any condition in the rectum. It is more a matter of consistency of the stool and the tone of the sphincter.

The fact that she had had constipation for many years does not mean anything. That statement could apply to a great many people who have nothing very seriously wrong with them.

Of the most importance I think is the fact that she had had cramp-like abdominal pain, each attack of which lasted 3 days. The pain was localized in the right upper quadrant which you would naturally associate with gallbladder disease. However, there is nothing more to substantiate the diagnosis of gallbladder disease, so I do not see how I can follow that up any further.

She noticed that her stools were blood-streaked. That again draws our attention to the colon. It does not say whether it was fresh blood but I presume it was, and I think that is most likely explained by the rectal examination, which showed a number of internal hemorrhoids.

Her past history we can say has nothing to do with the present illness.

The physical examination is of interest because I can see nothing of any great importance except that she was an obese woman, weighing 240 pounds at the age of 66. She had a moderate degree of hypertension for that age and for a woman of that size and build.

The x-ray showed marked arteriosclerosis. Abdominal examination was surprisingly negative. I doubt that moderate distention could have been detected even though it were present, because of the obesity. One would like to know whether there was thought to be any distention and also what the auscultatory signs over the abdomen were, but I see no mention of them. The vaginal examination and the rectal examination, aside from the hemorrhoids, were essentially negative. The proctoscopy was also negative, except for the lower end of the sigmoid,

where narrowing was noticed and a plug of mucus was seen occluding the passage.

Laboratory examination is of interest chiefly on account of the red cell count, which was perfectly normal, and a normal white count.

Differential Diagnosis

As to the question of diagnosis, it seems to me the lesion would most likely lie in the colon and I think the possibilities that are to be considered are carcinoma of the colon, diverticulitis, or perhaps some extrinsic cause of obstruction. I think I can rule out the latter very quickly as there is no evidence pointing toward it, either from abdominal, pelvic or rectal examinations. I think the diagnosis lies between the other two diseases, and in favor of diverticulitis I should say is the fact that there has been practically no bleeding. It is true that some people say that bleeding never occurs in diverticulitis. I think that probably is not true, as it occasionally does occur and there are figures that suggest that anywhere from 3 to 18 per cent may show bleeding. However, there certainly was very little bleeding in this case. It was noticed that there was narrowing of the lumen of the sigmoid which is perfectly consistent with diverticulitis, and it seems to me the presence of mucus occluding the lumen of the bowel would be rather in favor of a benign lesion, especially in the absence of any visible blood. Another point in favor of diverticulitis is the the fact that she is 66, weighs 240 pounds, and has a perfectly normal red count and hemoglobin. On the other hand in favor of carcinoma of the colon I concede the fact that she is 66 years of age and certainly might have malignant disease. One other possibility might be considered; that is, she might have a combination of both carcinoma of the colon and diverticulitis of the colon. It has been observed in a number of cases. I do not know the exact percentage, but the combination of these two diseases is shown.

A barium enema was done and perhaps Dr. Schatzki can help us.

Dr. Schatzki: There are two films here, one apparently with complete filling and one after emptying of the bowel. In the first film the barium is seen only in the rectum. I do not know the report of the examination but I think she did not expel the enema because the outside of the body looks perfectly clean and there is still a little barium in the rectum, so that I suppose there was an obstruction at the junction of the rectum and the sigmoid.

In regard to differential diagnosis between the two diseases that Dr. Linton has mentioned, the x-ray would help if barium actually reached the lesion. In this case we do not know that the barium reached the lesion since there might have been spasticity distal to the lesion, preventing the barium from reaching the lesion. There is certainly no tumor visible to explain the obstruction.

Another point that may be of interest is the fact that there is a slightly increased amount of gas in the descending colon and in the cecum, and no air whatever in the sigmoid.

Dr. E. L. Young: Is that a trickle of barium there?

Dr. Schatzki: Yes.

Dr. Young: That would suggest an actual narrowing of the lumen?

Dr. Schatzki: The lumen is definitely narrowed in this region.

Dr. Young: Could spasm there do that?

Dr. Schatzki: Yes.

Dr. Linton: That still leaves me with the decision to make, is this diverticulitis or carcinoma of the descending colon? I must admit I have not seen this degree of obstruction in simple diverticulitis of the colon and I think I would much rather favor the diagnosis of carcinoma of the colon with the possibility that it may be superimposed on the diverticulitis.

Clinical Discussion

Dr. E. Parker Hayden: This woman was a patient of mine in the Baker. She was very fat, weighing 240 pounds. She had complete obstruction as judged by a barium enema but I could not see any growth with the proctoscope, due to angulation of the sigmoid. No blood was seen at the end of the proctoscope. On the other hand she had a history of having passed some blood on occasions. We cleaned her out with castor oil without difficulty and operated after 3 days of preparation. The abdomen was not distended at the time of operation. There was a great deal of difficulty in the induction of anesthesia and nearly 2 hours were consumed before sufficient relaxation was achieved to permit satisfactory exposure of the tumor. When the tumor was finally seen and felt, it appeared to be entirely characteristic of carcinoma. Although originally a two-stage attack had been planned, the combination of her obesity and a short mesentery of the sigmoid made a loop colostomy difficult and it seemed best—in view of our definite impression that the mass was cancer—to go ahead in one stage. An anterior resection was performed, turning in the rectal stump and making an end colostomy. For 3 days postoperatively she seemed to do well, then suddenly on the fifth day she developed severe diarrhea through her colostomy, her temperature rose to 106° and she died that night.

Of course it is obvious, in retrospect, that a more conservative procedure was indicated. Had I thought it was diverticulitis, I would have done a colostomy only and not resected the mass. I did not think it was diverticulitis;

in fact it did not occur to me as being very likely. I did feel that she was a poor surgical risk but that here was a tumor which had better be taken out if possible.

PREOPERATIVE DIAGNOSIS

Carcinoma of the sigmoid.

DR. ROBERT R. LINTON'S DIAGNOSES

Carcinoma of the sigmoid.
Diverticulitis?

PATHOLOGIC DIAGNOSIS

Diverticulitis of the sigmoid.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: The specimen which we received after the operation was a segment of the lower end of the sigmoid. It was extraordinarily narrow, not even a lead pencil could be passed through it. At first glance the mucosa seemed almost normal, but as one pried it apart one could see the mouths of numerous diverticula some of which contained fecoliths. The wall of the bowel was very thick and fibrosed and the scar tissue extended out into the fat, making a hard indurated mass that undoubtedly would have felt essentially like malignant disease at the time of exploration.

Microscopic examination showed on the whole a very surprisingly small amount of inflammatory reaction. We have seen within the past year at least one other case of multiple diverticula with obstruction and very little inflammatory reaction. The obstruction seems to be due to a large extent to fibrosis—but of course it is not possible for a pathologist to rule spasm in or out.

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"ENSOL" THERAPY

THE first article a year ago on the Ensol treatment for cancer[1] reported on 29 cases. In the present bulletin of the Hendry-Connell Research Foundation of Kingston, Ontario, published in August, 1936, eight of the original 29 cases are reported to be alive, a survival of 27 per cent for ten months. The statement is made, "Several of these cases seem to have returned to normal health." A clinical evaluation is offered in this bulletin based on 488 patients treated with Ensol, of whom 382 received Ensol "alone," that is, no other treatment had been given within three months of the beginning of the Ensol therapy, and 106 in whom Ensol had been used with surgery, radium and x-ray in various combinations. The diagnosis of cancer had been confirmed by biopsy in 367 out of the 488 cases, or a total of 74 per cent. Of the 382 cases that had received no treatment for three months before the beginning of Ensol therapy, 180, or 47 per cent, had died. In a large proportion of the patients, relief of pain is stated to occur.

No definite statement is made as to the length of time that the patients had been followed. That it had been only a matter of a few months is apparent from the statement on page 25 of the bulletin that the survey had been completed on cases on whom treatment had been commenced prior to May 1, 1936, and that the returns had been compiled up to the end of July, 1936. It is also stated, "No recent case that has not been under treatment for *at least twelve weeks* has been included because it was felt that a *mature judgment* could not be formed as to the effects of Ensol." (The italics are ours.)

Apparently the substance is not harmful as in 16,000 individual doses there had been no generalized reaction and no evidence that normal tissue had been damaged. Included in the bulletin is a study of the cultural characteristics of *Bacillus histolyticus*.

The results, as presented thus far, are inconclusive and warrant continued skepticism of the value of this proposed type of cancer therapy.

REFERENCE

1. Canadian Medical Association Journal. 33:364, 1935.

CANNED FOODS

THE tin can has for years been a signpost of advancing civilization upon our frontiers, and today, when no frontiers are visible, it stands as a bulwark between the congested centers of population and the greatest fear of mass living — hunger. The opportunity which the Biblical Egyptians seized of filling their granaries in years of plenty against the years of want is transmitted to the modern housewife when she stocks her pantry shelves with canned goods against the time when supplies may temporarily fail or prices may rise. A knowledge of the preservation of food by heat sterilization, discovered by Appert in blockaded France in 1804, has driven the wolf from many a door, and for the majority of our population has helped to make the search for food something more than a hand-to-mouth existence.

The familiar and ubiquitous "tin can", derived from the English term "tin cannister", is actually about 98 per cent iron, being fabricated from rolled steel, covered with a thin coating of pure tin. Certain types of cans are coated with lacquer to preserve the natural flavor and color of various foods. The property of the can to preserve foods depends on heat sterilization and hermetic sealing, producing a vacuum inside the can when its contents are cooled.

Certain illusions concerning canned foods are dispelled in an interesting little booklet recently published by the American Can Company. One of these relates to the open can, in which many housewives are still unwilling to let food remain; fearing, no doubt, the deadly "pto-

maines''. Actually, due to the sterilization processes to which it has been subjected, the freshly opened can is the cleanest container in the kitchen in which to leave the food which came in it. Certain acid foods, after air is admitted, may absorb traces of metal and undergo changes in flavor, but their quality is otherwise unimpaired.

From the standpoint of the vitamins it has been demonstrated that vitamins A and G are not substantially affected by the canning process, that vitamin B is relatively stable in naturally acid foods, although less well retained in the less acid ones, and that vitamin C, the most labile of all the vitamins, is well retained in commercially canned foods, although subject to destruction by open pan methods of cooking which permit free contact with atmospheric oxygen. Our daily diet is not to be relied upon for our vitamin D needs, although there is evidence that canned marine products may contribute to it.

The can of late years has been given a tremendous impetus in infant feeding, due both to the increasing use of evaporated milk and the tremendous and justified popularity of the strained and specially prepared cereals, soups and vegetables. At the other end of the line, although not mentioned even by inference, is the conveniently packaged product of the brewmaster's art.

Canned foods as an important part of the civilized diet are with us to stay, until or unless some better method of food preservation is introduced. They are no longer a by-product as in the day when the farmer was asked what he did with his corn. ''We eat what we can,'' he replied, ''and what we can't we can.''

MATERNAL MORTALITY

In the *Westchester Medical Bulletin* of September, 1936, quotations appear taken from a report by Dr. James Quigley relating to Maternal Mortality as reported in Monroe County (Rochester, N. Y.) covering the years 1933, 1934, and 1935 as found in 17,868 births.

''. . . it can be seen that 40 deaths out of a total of 79 or approximately 50 per cent, were considered preventable. Of the 40 deaths responsibility was ascribed to the physician in 15 deaths or 37.5 per cent.''

Quotation in this article from another source relating to maternal mortality is: ''In the report of the committee of the New York Academy of Medicine 66 per cent of the deaths were considered preventable and the responsibility was charged to the medical attendant in 61 per cent.''

Further statistics on this subject are submitted by Dr. George W. Kosmak, Editor of the *Americal Journal of Obstetrics and Gynecology* in a paper read before the Thirty-Second Annual Congress on Medical Education, Medical Licensure and Hospitals, February 17, 1936.

The Westchester County Medical Society is continuing its investigations of this subject and has formulated recommendations for improved methods to be used in hospitals in the management of parturient cases. Dr. Kosmak declares that: ''Now, it has been found that the principal causes of death in childbirth are hemorrhage, shock, sepsis and toxemia, and for this group a preventable factor must be assumed if any degree of credit is to be given to the advances made in obstetric science and art. On what should the preventability factor be based? The answer may be found in the puerperal mortality studies of recent years to which reference has been made. In practically all these, if the particular death was considered avoidable, the blame is fixed on the doctors in a large proportion of cases.''

Farther on in his article, Dr. Kosmak emphasizes his conclusions and urges better training of physicians who are to practice obstetrics.

From these three sources and from unpublished expressions of opinions on many occasions there seems to be a call for action on the part of medical organizations. While it is encouraging to find that there exists a strong feeling that scientific medicine must be employed more generally in this field of practice, the question as to how to bring about the more general application of recognized preventive measures and better surgery is pertinent. May it not be necessary to inaugurate specific studies of maternal deaths more generally by organized medicine rather than to wait for a spontaneous evolution by general practitioners?

Specialists in obstetrics will of course be in the vanguard of progress and will in the future serve larger proportions of parturient patients, but why not impress on the family doctor that he must prepare himself for first-class service or refer his patients to competent practitioners?

If every maternal death were subjected to a critical review and an authorized committee discussed the facts with physicians in charge, better results might follow.

THE COUNCIL MEETING OF THE MASSACHUSETTS MEDICAL SOCIETY

On October 7 an unusually large number of Councilors were in attendance at the Stated Meeting.

The routine business was voluminous, but was transacted promptly, so that with the earlier meeting hour there was no need of a recess for luncheon.

Two matters, of especial interest to the profession, were presented. The Committee on

Medical Education and Medical Diplomas, through its Chairman, Dr. Reginald Fitz, reported the receipt of a letter from an official of the Middlesex College School of Medicine in which the suggestion was made that the good offices of the Massachusetts Medical Society in plans for raising the standards of medical education by that institution would be appreciated. The sentiment of the meeting seemed to be friendly and co-operative.

The other matter of especial interest was presented by Dr. Tighe of Lowell. This related to the published statement in *The New England Journal of Medicine* setting forth that a Fellow of the Society had been sued for alleged conspiracy in signing papers submitted to a court recommending study of the behavior of a certain person by a State Hospital for mental illness.

The outcome of this case will be watched because, if it is found that physicians who sign commitment papers in cases of suspected mental disorders, are subject to such suits, there should be some safeguards for those who are called upon to exercise this important function.

Reference to these two matters must not be construed as adequate in dealing with this meeting, because every item of business should be carefully considered by all members and it is hoped that this brief statement will lead to careful reading of the Proceedings which will be published as soon as prepared by the Secretary, Dr. Begg.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

BRYAN, WILLIAM A. M.D. George Washington University School of Medicine 1908. Superintendent, Worcester State Hospital. His subject is "Relationship of Psychiatry to Medicine." Page 693. Address: Worcester State Hospital, Worcester, Mass.

PERKIN, H. J. M.A. University of Toronto. Research Fellow in Biochemistry, Lahey Clinic. Address: 605 Commonwealth Avenue, Boston, Mass. Associated with him is

HURXTHAL, L. M. M.D. Harvard University Medical School 1923. Senior Physician, Lahey Clinic. Physician, New England Baptist and New England Deaconess Hospitals. Address: 605 Commonwealth Avenue, Boston, Mass. Their subject is "The Blood Iodine Level, Before and After Subtotal Thyroidectomy for Hyperthyroidism." Page 698.

SMITH, RICHARD M. Sc.D., M.D. Harvard University Medical School 1907. Assistant Professor in Pediatrics and Child Hygiene, Harvard University Medical School and Harvard School of Public Health. His subject is "Acute Gastrointestinal Disease in Infants." Page 701. Address: 66 Commonwealth Avenue, Boston, Mass.

LADD, WILLIAM E. A.B., M.D. Harvard University Medical School 1906. F.A.C.S. Chief of Surgical Service, Children's Hospital, Boston. Clinical Professor of Surgery, Harvard University Medical School. His subject is "Surgical Diseases of the Alimentary Tract in Infants." Page 705. Address: 300 Longwood Avenue, Boston, Mass.

CHRISTIAN, HENRY A. A.M., LL.D., Sc.D. (Hon.), M.D. Johns Hopkins University School of Medicine 1900. Hersey Professor of the Theory and Practice of Physic, Harvard University Medical School. Physician-in-Chief, Peter Bent Brigham Hospital, Boston. His subject is "Diuretics and What They Do." Page 709. Address: Peter Bent Brigham Hospital, Boston, Mass.

EMERY, EDWARD S., JR. A.B., M.D. Harvard University Medical School 1920. Instructor in Medicine, Harvard University Medical School. Associate in Medicine, Peter Bent Brigham Hospital. His subject is "Progress in Gastro-Enterology for 1935." Page 712. Address: 319 Longwood Avenue, Boston, Mass.

The Massachusetts Medical Society

FOURTH ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following sessions have been arranged by the Committee for the week beginning October 19:

Barnstable

Sunday, October 25, at 4:00 p. m., at the Cape Cod Hospital, Hyannis. Subject: Blood Diseases: The Hemoglobin and Red Blood Cells in Relation to Disease. Instructor: W. B. Castle. John I. B. Vail, Chairman.

Berkshire

Thursday, October 22, at 4:30 p. m., at the House of Mercy Hospital, Pittsfield. Subject: Neurological Surgery: The Signs and Symptoms of the Common Brain Lesions—Organic and Traumatic. Instructor: J. S. Hodgson. Melvin H. Walker, Jr., Chairman.

Essex South

Tuesday, October 20, at 4:00 p. m., at the Salem Hospital, Salem. Subject: Diabetes: General Plan of Treatment in *Uncomplicated* Cases; Diet; Insulin (Regular and Protamine); Exercise. In-

structor: Reginald Fitz. Walter G. Phippen, Chairman.

Franklin

Wednesday, October 21, at 8:00 p. m., at the Franklin County Public Hospital, Greenfield. Subject: Cancer of the Genito-Urinary Tract. Instructor: J. D. Barney. Halbert G. Stetson, Chairman.

Hampden

Thursday, October 22, at 4:00 p. m., at the Academy of Medicine, Professional Building, 20 Maple Street, Springfield, and at 8:30 p. m. in the Outpatient Department of the Skinner Clinic, Holyoke Hospital, Holyoke. Subject: *Complications* of *Diabetes* and Their Treatment. Coma; Insulin Reactions; Surgery (Gangrene, Carbuncle, etc.); Marriage and Pregnancy; Tuberculosis and Heart Disease. Instructor: H. F. Root. George L. Schadt and George D. Henderson, Chairmen.

Hampshire

Wednesday, October 21, at 4:15 p. m., in the Nurses Home of the Cooley Dickinson Hospital, Northampton. Subject: The Prognosis of Heart Disease. Instructor: Sylvester McGinn. Robert B. Brigham, Chairman.

Middlesex East

Tuesday, October 20, at 4:00 p. m., at the Melrose Hospital, Melrose. Subject: Neurological Surgery: The Signs and Symptoms of the Common Brain Lesions—Organic and Traumatic. Instructor: W. R. Wegner. Joseph H. Fay, Chairman.

Norfolk

Friday, October 23, at 8:30 p. m., at the Norwood Hospital, Norwood. Subject: Heart Disease: Treatment of Cardiovascular Emergencies. Instructor: B. E. Hamilton. Hugo B. C. Riemer, Chairman.

Worcester (Milford Section)

Thursday, October 22, at 8:30 p. m., in the Nurses Home of the Milford Hospital, Milford. Subject: *Complications* of *Diabetes* and Their Treatment. Coma; Insulin Reactions; Surgery (Gangrene, Carbuncle, etc.); Marriage and Pregnancy; Tuberculosis and Heart Disease. Instructor: Priscilla White. Joseph Ashkins, Sub-Chairman

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Applicants for Examination by Censors

The following: Dr. Lawrence T. Mutty, 42 Union Street, Manchester, and Dr. Thomas Francis Quinn, 548 Western Avenue, Lynn, have each received letters approving their application for membership in the Massachusetts Medical Society, from five doctors of their respective towns.

R. E. Stone, M.D., *Secretary.*

MISCELLANY

VOCATIONAL REHABILITATION AND WORKMEN'S COMPENSATION

There is little or no therapeutic benefit in cash settlements paid to injured workmen who have traumatic neuroses, according to an investigation made recently by Carl Norcross, Ph.D., of the Rehabilitation Division of the New York State Department of Education. Results of the investigation have been published under the title "Vocational Rehabilitation and Workmen's Compensation" and the report is a follow-up study of 322 workmen's compensation cases throughout New York, which were closed by a lump-sum settlement of $1000 or more.

It has been generally accepted in both medical and workmen's compensation circles throughout the country, says the report, that a cash award would help to cure a neurosis. "A careful investigation made a year or more after the settlements has convinced us that the value of a cash award is vastly overrated," writes the author. "It is the settlement of the case, the actual ending of the litigation, which is of value. Whether the final compensation award is paid in one lump or extended through a number of installments makes little difference to the claimant's condition."

The investigation disclosed that 16 per cent of the men had lost a large share of their compensation through unwise expenditures. The men who had no losses were found to have dissipated their funds much more rapidly than they would have under an installment system. Both because there was found to be a wastage of compensation funds, and because there appeared to be no therapy in the settlements, the author has recommended that lump-sum settlements be discontinued.

Dr. Norcross makes a number of recommendations for improving the handling of neurotic cases in the workmen's compensation rooms. He urges that cases be given a more prompt and careful handling. The average neurotic case is open in the workmen's compensation division for nearly three and one-half years, it is said, and much of the delay is unnecessary. The author states that neurotic conditions grow as cases are delayed. He also points out the dangerous policy of permitting claimants to read their own medical reports, or to be present when physicians are testifying, especially in contested cases where there is a difference of opinion.

In New York a compensation case theoretically may always be reopened. The report suggests that it is a poor policy to let neurotic claimants know that, when their money is spent, they may try to reopen their cases. The author believes that one of the evils of the existing New York system in nonschedule cases is that claimants must be willing to accept a lump-sum settlement. After a fair offer is made, the neurotic claimant may procrastinate indefinitely by refusing such a settlement. Thus the case is delayed, and the patient's mental condition

may become worse. The remedy suggested by the report is that the referee, acting on competent medical advice, fix a fair settlement and close the case, with the award being paid in bi-weekly installments.

To overcome any prejudice the claimant may have toward the insurance company, it is suggested that the money be paid to a State administered trust fund, which already exists in New York. The carrier could close the case on its books, and the claimant could be told his case is definitely closed but that he would get all his money, regardless of his state of health. The patient would not have to remain sick to get his award.

Provision is made for permitting the claimant to get an advance on his compensation for any necessary purpose, including rehabilitating himself on a farm or in a small business.

Copies of the report may be secured through the publisher, The Rehabilitation Clinic, 28 East 21st Street, New York City. Price $1.

BUREAU OF HUMAN HEREDITY

The object of this Bureau is collection on as wide a scale as possible of material dealing with human Genetics. Later, the tasks of analysis of material and distribution of the information available will be added.

The Bureau is directed by a Council representing medical and scientific bodies in Great Britain. It is affiliated with the International Human Heredity Committee, which ensures co-operation in all areas where research is proceeding.

The Council would be grateful to receive all available material from institutions and individuals, furnishing well-authenticated data on the transmission of human traits whatever these may be. Pedigrees are particularly desired; twin studies and statistical researches are also relevant. As research workers and others who send in material may in some cases wish to retain the sole right of publication (or copyright) those who so desire are asked to accompany their material with a statement to that effect.

Material should be given with all available details in regard to source, diagnostic symptoms and the name and address of the person or persons who vouch for accuracy. All such details will be regarded as strictly confidential.

Reprints of published work would be most acceptable. Further, many authors when publishing material may also have collected a number of pedigrees which they have been unable to reproduce in detail. It is the object of the Council that such records, by being included in the Clearing House, should not be lost.

Those wishing for a copy of the Standard International Pedigree Symbols may obtain one from the office.

Announcements in regard to the services undertaken by the Bureau will be published from time to time.

Chairman: R. Ruggles Gates.

Executive Committee: R. A. Fisher, J. B. S. Haldane, E. A. Cockayne, J. A. Fraser Roberts, L. E. Halsey (Hon. Treasurer), C. B. S. Hodson (Hon. Gen. Secretary).

115 Gower Street, London, W.C.1, England.

MAINE NEWS

The President's Page*

To the Members of the Maine Medical Association:

According to Article II of our Constitution, "The purposes of this Association are to promote the science and art of medicine, the protection of public health and the betterment of the medical profession." These few phrases state concisely and definitely our whole creed. There is no need for elaboration. It only remains for us to keep these pregnant phrases ever in mind. Perhaps the first portion is the most important, for the rest, the protection of public health and the betterment of the medical profession, depend very largely upon our efforts to further the science and art of medicine.

If we, as physicians, are to follow the precepts of our Constitution we must devote a certain amount of our time to the study of our profession, keeping abreast of its most progressive thought. We may do this by reading scientific journals, postgraduate study, attendance at clinics and by an active participation in our national, state and county medical meetings. All these are important, the last no less than the others. Yet, possibly because this is so readily available, it is too often neglected. If every physician would regularly attend and take part in each meeting of his county and state association, demanding by his very presence that these meetings be of sufficiently high standard to merit his interest, what a power for good would our Association become.

It is the desire of your officers this year to endeavor to carry out the purpose of the Association as set forth in Article II of our Constitution. We hope to increase the interest and the value in meetings of the constituent County Associations. Regular set dates for meetings, more frequent meetings in many cases, carefully selected and prepared programs, all these should tend to a better attendance and consequently a better society. We hope to make our State Journal a state-wide medium of publication of medical material, publishing not only papers read at the annual meeting but also those presented at county meetings. If a paper is worthy of presentation before one county society, it should be available, through the *Journal*, to the whole Association. The success of this depends largely upon the county secretaries. If they will co-operate with the members

*Reprinted from the Maine Medical Journal, 27:190, 1936.

of the Editorial Board by turning over to them selected papers from their meetings, we can make our *Journal* represent the Transactions of the County Associations as well as the State.

Four years ago, President Kershner inaugurated the Fall Clinical Session. This was perhaps the outstanding achievement of one of the most brilliant administrations we have ever had. Dr. Kershner contributed a great deal to the Association but nothing of greater value than this opportunity for a short, concise review of clinical problems, handled as we have to handle them ourselves. The sessions at Bangor, Portland and Lewiston fully justified Dr. Kershner's idea. It is hoped and expected that the session next month in Waterville will do likewise. It largely depends upon you men. A well-prepared program will be presented. A good attendance is all that is necessary to make the session a success. Come and see how your confrere, situated as you are, handles his problems. Discuss it freely, criticize if you will, approve if you feel so disposed; but come and take an active part. Both you and your confrere will benefit by it. It is just one more way to help make our Association stronger and to further "promote the science and art of medicine". And it doesn't do us a bit of harm to get together once in a while to socialize a little, as only doctors can, at the close of a day's meeting.

FREDERICK T. HILL, M.D.

On Sept. 13, 1936, a meeting of the Editorial Board of the *Maine Medical Journal* was held at the Penobscot Valley Country Club. Owing to the recent resignation of Dr. E. W. Gehring of Portland as Editor-in-Chief, a change in the management of the *Journal* was voted upon. It was voted that the *Journal* should be run by active participation of the entire Editorial Board with Dr. Frank H. Jackson, of Houlton, as Acting Chairman.

It was voted that the policy of the *Journal* should be one in which the aim should be to publish purely State of Maine material, with the exception of those papers read before the Maine Medical Association's annual meeting or before county medical societies. The effort was to be made to encourage the writing of papers for the county medical societies, especially by the younger men.

The annual meeting of the Piscataquis County Medical Society was held Sept. 17, 1936, at the Piscataquis Valley Country Club. Dr. Edward H. Risley of Waterville read a paper on "The Treatment of Certain Postoperative Complications," and Dr. F. T. Hill, President of the Maine Medical Association, outlined briefly a few of the problems of the Association for the coming year. He spoke of the re-organization of the *Journal* and the desire to induce more men to contribute scientific papers to the *Journal*. He also emphasized the importance of the county medical society as the strongest factor in building a strong state society and urged more frequent stated meetings of the county societies with programs planned at least two months before the time of meeting.

CORRESPONDENCE

THE HUMOR OF DR. OLIVER WENDELL HOLMES

Editor, *New England Journal of Medicine*,

Apropos of the address of Dr. Robert M. Green on Dr. Oliver Wendell Holmes, published in your issue of September 10, the following anecdote, never before published, may be of interest to your readers:

When Holmes was professor in Harvard Medical School, there was among his students a young man named L. W. Clapp, afterward a very skillful physician in Pawtucket, Rhode Island.

Dr. Clapp died many years ago, but the incident I am relating was told me by him so there is no doubt of its truth.

Clapp and another medical student were going to Holmes' lecture room one winter day to attend the lecture. They went afoot and their path led at one place over a vacant lot which was being filled in, to bring it up to a level with surrounding land. As they were passing through this lot, Clapp's companion noticed a couple of turkey legs among the earth dumped on the lot. The name of this companion I do not remember, but Dr. Clapp called him Jack.

To the astonishment of Clapp, Jack picked up the turkey legs, wiped them carefully and put them in his pocket.

"What in the world are you going to do with turkey legs?" asked Clapp. Jack replied laconically, "Wait and see."

The class assembled as usual in the lecture room and Dr. Holmes began his lecture. He had proceeded about five minutes when, to his amazement, an object hurtled through the air and landed on his desk. It was a turkey leg. Dr. Holmes paused a moment, and when the merriment subsided, resumed his lecture.

A few minutes later another turkey leg flew over the heads of the students in front and almost hit the professor. This time he again waited until the room was quiet, and then, with the well-known twinkle in his eyes, recited this quotation from Young's "Night Thoughts"—

"Insatiate Archer, would not one suffice?"

A more felicitous response can scarcely be imagined, and, needless to say, the class fully appreciated the brilliancy of the quotation.

Respectfully yours,
JAMES L. JENKS.

255 Main Street, Pawtucket, R. I.,
September 25, 1936.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Illinois,
September 28, 1936.

Managing Editor,
New England Journal of Medicine,

In addition to the articles enumerated in our letter of September 1 the following have been accepted:

International Vitamin Corporation
I.V.C. Halibut Liver Oil, Plain
Capsules I.V.C. Halibut Liver Oil, Plain, 3 minims
I.V.C. Halibut Liver Oil Fortified with Natural Vitamin D
Capsules I.V.C. Halibut Liver Oil with Vitamin D Concentrate in Neutral Oil, 3 minims

Lederle Laboratories, Inc.
Pollen Antigen-Lederle, Mixed Grasses
Concentrated Pollen Antigen-Lederle, Mixed Grasses
Scarlet Fever Streptococcus Immunizing Toxin-Lederle, one 2 cc. vial containing 80,000 to 100,000 skin test doses

United States Standard Products Co.
Ampuls Solution Caffeine Sodio-Benzoate 7½ grains, 2 cc.

The following products have been accepted for inclusion in the List of Articles and Brands Accepted by the Council But Not Described in N.N.R. (New and Nonofficial Remedies, 1936, p. 471):

Lederle Laboratories, Inc.
Glycerinated Allergenic Extracts (Diagnostic)-Lederle (Bee, House Fly, Mosquito and Sand Fly)

Yours sincerely,
PAUL NICHOLAS LEECH, *Secretary,*
Council on Pharmacy and Chemistry.

RECENT DEATH

BARRY—EMMETT WILLIAM BARRY, M.D., of Whitinsville, Massachusetts, died October 6, 1936, after a short illness.

Dr. Barry was born in 1871. He graduated from the College of Physicians and Surgeons of Baltimore in 1897. He practiced for a year in Uxbridge and then moved to Whitinsville where he practiced up to the time of his death.

Dr. Barry was a member of the Knights of Columbus, the Hibernians and the United Woodmen. He was a Fellow of the Massachusetts Medical Society. A widow, a son, a daughter, and two brothers survive him.

NOTICES

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, October 22, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Soma Weiss, Associate Professor of Medicine, Harvard Medical School, and Director 2nd and 4th Medical Services (Harvard), Boston City Hospital, will give a medical clinic. To it are cordially invited practitioners and medical students.

UNITED STATES CIVIL SERVICE EXAMINATIONS

PRINCIPAL INDUSTRIAL TOXICOLOGIST (HEAVY METALS), $5,600 A YEAR

TREASURY DEPARTMENT, PUBLIC HEALTH SERVICE

Applications must be on file with the United States Civil Service Commission at Washington, D. C., not later than November 2, 1936.

The United States Civil Service Commission announces an open competitive examination for the position named above. Vacancies in this position in Washington, D. C., and in the field, and in positions requiring similar qualifications will be filled from this examination, unless it is found in the interest of the service to fill any vacancy by reinstatement, transfer, or promotion. The salary named above is subject to a deduction of 3½ per cent toward a retirement annuity.

Duties.—To inaugurate, plan, and direct investigations upon the toxicology of heavy metals, and to furnish for executive action expert or critical advice upon the industrial toxicology of heavy metals.

Basis of Ratings. — Competitors will not be required to report for examination at any place, but will be rated on their education and experience on a scale of 100, such ratings being based upon competitors' sworn statements in their applications and upon corroborative evidence.

SENIOR MEDICAL TECHNICIAN (BONE PATHOLOGY), $2,000 A YEAR, CHILDREN'S BUREAU, DEPARTMENT OF LABOR

Applications must be on file with the United States Civil Service Commission at Washington, D. C., not later than November 2, 1936.

The United States Civil Service Commission announces an open competitive examination for the position named above. A vacancy in this position in the field and vacancies occurring in positions requiring similar qualifications will be filled from this examination, unless it is found in the interest of the service to fill any vacancy by reinstatement, transfer, or promotion. The salary named above is subject to a deduction of 3½ per cent toward a retirement annuity.

A vacancy exists in the Children's Bureau, Department of Labor, Baltimore, Md.

Duties.—To study the histologic and pathologic aspects of bones; to prepare, or supervise the prepara-

tion of, sections of bones; to describe the microscopic findings, and to analyze and evaluate the results.

THE APPOINTMENT OF DR. SAMUEL J. BECK

Dr. Samuel J. Beck has been appointed in charge of the Psychology Laboratory in the Department of Psychiatry at the Michael Reese Hospital. Dr. Beck was formerly with the Department of Psychiatry of the Harvard Medical School and the Psychopathic Hospital, Boston, Mass., where he was a research associate.

Dr. Beck holds a Ph.D. degree from Columbia University and has been a Rockefeller Fellow in Psychiatry, studying abroad. He is the associate editor of the *American Journal of Orthopsychiatry*.

ANNOUNCEMENTS

EDWIN B. SEELYE, M.D., announces the opening of an office associated with WALTER C. SEELYE, M.D., in the Slater Building, 390 Main Street, Worcester, Massachusetts. Room 447.

FRANKLIN E. CAMPBELL, JR., M.D., announces his transfer from the U. S. Marine Hospital, Chelsea, Mass., to the U. S. Marine Hospital, Seattle, Washington.

MARCUS W. BERMAN, M.D., announces the opening of an office at 304 Belgrade Avenue, West Roxbury, Mass.

REPORTS AND NOTICES OF MEETINGS

FAULKNER HOSPITAL CLINICAL MEETING

The usual monthly clinical meeting was held at the Faulkner Hospital on Thursday afternoon, October 1, at 5:00 p. m. Among the interesting cases that had come to autopsy during the summer months, two were selected for discussion.

One was the case of a woman, 55 years of age, who entered the hospital with the complaint of severe headaches, mental confusion and periods of semi-consciousness, from which, however, she could be aroused. A cerebral tumor or some vascular accident in the brain were the two most likely diagnoses. Some pathology within the central nervous system was supported by the presence of increased cells in the spinal fluid and a xanthochromic spinal fluid. Finally by ventriculogram a tumor was located in the right posterior temporal occipital region and this diagnosis was confirmed at operation, at which an inoperable tumor was found, that at autopsy proved to be a glioma. One of the interesting points in this case was the fact that just a month before the onset of the symptoms definitely pointing toward the brain, this patient had been operated on for typical symptoms of hyperthyroidism, and was relieved of all of her symptoms so that she felt herself well on leaving the hospital, about two weeks before the severe headaches developed. The symptoms of hyperthyroidism did not begin until January, 1936. At the time of the development of these symptoms, there was an occasional headache and some tremor of the feet. It is interesting to speculate if there could be any relation between this glioma of the brain and the typical picture of hyperthyroidism.

The other case presented was that of a man 22 years of age, who had had rheumatic fever ten years before, at which time it was noted that a valvular disease of the heart had developed. Only 5 weeks before death did symptoms arise, and these consisted in feeling poorly and feverish. The fact that he was running a fever was established only 3½ weeks before his death. He ran a continued fever. There was some looseness of the bowels. There was aching in the knees and calves of the legs and a loss in weight of about 10 pounds. On physical examination, there were numerous petechial spots. There were evidences of chronic cardiac valvular disease, consisting in a presystolic thrill and a presystolic murmur with a snapping first sound at the apex and a systolic murmur heard all over the precordium. The electrocardiogram showed no evidence of myocardial involvement. The clinical pathology showed some albumin and casts in the urine, a leukocytosis of 22,000 with 80 per cent of polynuclear cells and a steadily increasing diminution in red count starting at 4,500,000 two weeks before death and dropping to 3,350,000 a few days later. A blood culture showed a positive growth which was reported as *Streptococcus viridans*. During the last few days of life, he developed a panophthalmitis in one eye and signs of meningeal irritation, which were thought to be due to invasion of the meninges with bacteria or to numerous embolic phenomena in the meninges. It was thought to be a typical case of *Streptococcus viridans* septicemia with involvement of the diseased cardiac valves, but it was considered peculiar in view of the short duration of its course. At autopsy, a chronically diseased mitral valve was found with vegetations upon it. Also embolic areas, some developing into abscesses were found at the cortex of the brain extending to the meninges. The kidneys showed embolic phenomena with beginning abscess formation. There were some beginning abscesses in the lungs. Cultures, however, from the heart's blood, vegetations on the valves and the involved eye all showed an organism which turned out to be a pneumococcus, Type V, and it was felt without doubt that this was the cause of the disease and that undoubtedly the organism which had been reported as a *Streptococcus viridans* was this pneumococcus which had perhaps been modified somewhat in its character as it circulated through the blood stream.

Following the presentation of these two cases, Dr. Burton E. Hamilton discussed "The Treatment of

Bacterial Endocarditis", including streptococcus, pneumococcus and other types. He called attention to the importance of being absolutely sure of the type of organism before reporting cures of a specific type of infection, using the case just reported to illustrate how, had it not been for the bacteriologic studies at the postmortem examination, the case would have been erroneously recorded as one of *Streptococcus viridans* infection.

Apparently bacterial endocarditis is on the increase according to the statistics. Available figures suggest that 5 per cent of all cases of rheumatic fever with involvement of the valves of the heart die with bacterial endocarditis.

Dr. Hamilton brought out the point which is still under discussion, whether the *Streptococcus viridans* is the cause of rheumatic fever. If so, the eventual invasion of the blood stream with this organism, and its location upon the diseased valves of old rheumatic fever may be a condition similar to the development of miliary tuberculosis originating from some long-continued focus of tuberculous infection. He raised the question of whether bacterial endocarditis can develop upon healthy valves and, instead of continuing on to a fatal termination, heal up. There is some evidence to support this idea. Usually with bacterial endocarditis the myocardium is not involved although in rheumatic fever it usually is. He mentioned the interesting case of a bacterial growth developing upon the site of an arteriovenous aneurysm which resulted in cure by the excision of the diseased area.

In regard to treatment, the bactericidal substances which have been suggested were dismissed without comment on the ground that any substance strong enough to kill off these bacteria would be detrimental to the host.

It was brought out in the discussion that hyperpyrexia is not of value in these conditions because the organisms are resistant to higher temperature than the body can stand. He considered that attempts to develop an immune sera for streptococcus infections and pneumococcus infections would probably be of no value because the organisms are already existing in the circulation and presumably have produced as much reaction as possible and do not succeed in causing appreciable trouble until they find a haven in which they can grow, apparently protected from the antibodies in the blood stream. Therefore, little can be hoped for by introducing more antibodies, in view of the fact that these organisms have developed in a position where they are apparently protected from the serum. In cases of meningococcus and gonococcus endocarditis, the value of antisera must be kept in mind, because favorable results have been reported, but the likelihood of real benefit by this treatment is small.

He called attention to the instances of animals, in which antipneumococcus serum has been developed, dying of pneumococcus septicemia. He brought up the question of the relation between oral sepsis due to *Streptococcus viridans* and the development of bacterial endocarditis from this source. The evidence suggests that there may be such a relation, and therefore, as a prophylactic procedure, these organisms should be eliminated from lesions about the teeth.

In summary he felt that attempts at specific treatment against bacterial endocarditis should not, because of the disturbance and expense associated therewith, be undertaken with the hope of real benefit unless circumstances made experimental study seem justifiable.

SUFFOLK DISTRICT MEDICAL SOCIETY

Censors' Meeting

The Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 Fenway, Thursday, November 5, 1936, at 4:00 o'clock.

Candidates should make personal application to the Secretary, and present their medical diplomas at least one week before the examination.

Charles C. Lund, M.D., *Secretary.*

319 Longwood Avenue, Boston.

1936-1937 Schedule

October 28, 1936—Stated Meeting. Boston Medical Library. 8:15 p. m.

"Report on a Study of Maternal Mortality in Boston Made by the Obstetrical Society of Boston and the Boston City Department of Health." Dr. Robert L. DeNormandie.

Discussion: Dr. Charles F. Wilinsky and Dr. John T. Williams.

(Final notice of this meeting. No postcards will be sent out.)

November 18, 1936 — Boston Medical Library. 8:15 p. m.

"Hydrocarbons and Cancer." Dr. M. J. Shear—U.S.P.H. Service.

"Cancer Research. Recent Advances in Our Knowledge of Cancer." Dr. J. C. Aub.

Discussion: Dr. J. W. Schereschewsky—U.S.P.H. Service and Dr. R. B. Greenough.

January 27, 1937 — Boston Medical Library. 8:15 p. m. Joint Meeting with the Boston Medical Library.

"Anthropology." Dr. Carleton S. Coon.

March 31, 1937—Boston Medical Library. 8:15 p. m.

"Social Insurance—It Affects the Medical Profession." Dr. Charles E. Mongan.

Discussion: Dr. Channing Frothingham.

April 28, 1937 — Annual Meeting. Boston Medical Library. 8:15 p. m.

"Problems in Surgical Diagnosis." Dr. Howard M. Clute.

Conrad Wesselhoeft, M.D., *President,*
Charles C. Lund, M.D., *Secretary.*

NORFOLK DISTRICT MEDICAL SOCIETY

A stated meeting of the Society will be held in St. Elizabeth's Hospital, Brighton, October 27, 1936, at 8:00 p. m. Tel. Sta. 7000.

Business: 8:00 p. m.

Communications: 8:15 p. m.

1. Indication for Spinal Fusion—Dr. Thomas F. Broderick.
2. Recent Advances in the Treatment of Pruritus Vulvae—Dr. Charles J. Kickham.
3. Medical Aspects of Gallbladder Disease—Dr. John J. Whoriskey.
4. Surgical Aspects of Gallbladder Disease—Dr. Joseph Stanton.
5. Radiological Aspects of Yeast Infection of the Lungs—Dr. Thomas R. Healy.
6. The Treatment of Pneumonia—Dr. John F. Casey.

Twelve minutes will be allotted to each speaker. Discussion will be postponed until after all communications have been completed.

FRANK S. CRUICKSHANK, M.D., *Secretary.*
1247 Beacon Street, Brookline.

Postgraduate Extension Courses: These lectures will begin on October 16 and will continue through November 20. They will be given in the Norwood Hospital on stated Fridays at 8:30 p. m. Full particulars may be obtained from the District Chairman, Dr. Hugo B. C. Riemer, 128 Newbury Street, Boston, Tel. Kenmore 2826.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance), Tuesday evening, October 27, at 8:15 p. m.

PROGRAM

Presentation of Cases.

Cases of Attempted Suicide in a General Hospital. By Merrill Moore, M.D., Associate in Psychiatry, Harvard Medical School.

Medical students and physicians are cordially invited to attend.

MARSHALL N. FULTON, M.D., *Secretary.*

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

The Worcester North District Medical Society will hold a regular quarterly meeting at the state colony in Gardner at 4:30 p. m., Wednesday, October 28. The speaker will be Dr. Harvard H. Crabtree of Boston.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, OCTOBER 19, 1936

Tuesday, October 20—

All-day meeting. Academy of Physical Medicine. Hotel Statler, Boston.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Treatment of Gastro-Intestinal Disorders in Infants. Dr. Francis McDonald.

9:30 a. m. Massachusetts General Hospital. Thoracic Clinic. Ether Dome.

11:30 a. m. Massachusetts General Hospital. Eye Nerve Conference. Out-Patient Department.

*12 m. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

12 m. Boston Dispensary. Luncheon meeting of the Clinical Staff.

Wednesday, October 21—

All-day meeting. Academy of Physical Medicine. Hotel Statler, Boston.

8 a. m. Massachusetts General Hospital. Orthopedic Rounds.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

†12 m. Clinico-Pathological Conference. Children's Hospital Amphitheatre.

Thursday, October 22—

All-day meeting. Academy of Physical Medicine. Hotel Statler, Boston.

9 a. m. Massachusetts General Hospital. Surgical Grand Rounds. Amphitheatre.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street. Social Service Case Presentation. Miss E. C. Canterbury.

9:15 a. m. Massachusetts General Hospital. Neurological Conference. Ether Dome.

11 a. m. Massachusetts General Hospital. Medical Grand Rounds. Ether Dome.

12 m. Massachusetts General Hospital. Clinico-Pathological Conference.

*3:30 p. m. Medical Clinic, Peter Bent Brigham Hospital.

5:15 p. m. Massachusetts Medical Benevolent Society. Boston Medical Library, 8 Fenway.

Friday, October 23—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Recent Work in Epilepsy and Migraine. Dr. William G. Lennox.

10:30 a. m. Massachusetts General Hospital. Fracture Rounds.

12 m. Massachusetts General Hospital. Urological Conference. Out-Patient Department.

Saturday, October 24—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

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*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

October 16—The Celebration of the Use of Ether. Massachusetts General Hospital. See page 689, issue of October 8.

October 18—Special Service for Physicians and Medical Students. See page 691, issue of October 8.

October 19-23—Clinical Congress of the American College of Surgeons, Philadelphia.

October 19-31—1936 Graduate Fortnight of the New York Academy of Medicine. See page 1221, issue of June 11.

October 20—South End Medical Club. See page 692, issue of October 8.

October 20-22—Academy of Physical Medicine, Annual Meeting, Hotel Statler, Boston. See page 692, issue of October 8.

October 20-23—The American Public Health Association. See page 1226, issue of June 11.

October 22—Massachusetts Medical Benevolent Society, Annual Meeting. See page 691, issue of October 8.

October 22—Medical Clinic at the Peter Bent Brigham Hospital. See page 738.

October 27—Harvard Medical Society. See notice elsewhere on this page.

November 12—Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

November 16—One hundredth anniversary of the founding of the Army Medical Library, 7th Street and Independence Avenue, S. W., Washington, D. C.

December 3-5—Annual Conference of the National Society for the Prevention of Blindness, Columbus, Ohio.

March 30 - April 2, 1937—First International Conference on Fever Therapy. Postponement notice. See page 52, issue of July 2.

April 21-24, 1937—American Society for Experimental Pathology. See page 1075, issue of May 21.

DISTRICT MEDICAL SOCIETIES

FRANKLIN DISTRICT MEDICAL SOCIETY

Will meet at the Weldon in Greenfield at 11 a. m. the second Tuesdays of November, January, March and May.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

HAMPDEN DISTRICT MEDICAL SOCIETY

October 20—Regular Fall Meeting at the Springfield Academy of Medicine, 20 Maple Street, Springfield, at 4:15 p. m.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

November 18—Bear Hill Golf Club, Stoneham.

January 13, 1937—Bear Hill Golf Club, Stoneham.

March 16, 1937—Danvers State Hospital, Danvers.

May 11, 1937—Bear Hill Golf Club, Stoneham.

KENNETH L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

October 27—See page 741.

November 24—8:15 p. m. The Beth Israel Hospital. Communications and Case Presentations by the Staff. Principal subject—Cardiology. Details of program to be announced.

January 19, 1937—8:15 p. m. The Peter Bent Brigham Hospital. Communications and Case Presentations by the Staff. Suggested title—"Abdominal Pain from the Medical and Surgical Standpoint." Details of program to be announced.

February 23, 1937—Time, place and details of program to be announced.

March 30, 1937—8:15 p. m. New England Deaconess Hospital. A Symposium on Diabetes entitled "A Survey of the Diabetic Work of the George F. Baker Clinic in the New England Deaconess Hospital." Communications and Case Presentations by the Staff. Drs. Elliott P. Joslin, Howard F. Root, Priscilla White, Alexander Marble and Allen P. Joslin.

May, 1937—Annual Meeting. Details to be announced.

The Secretary on behalf of the Society and its Executive Committee desires to express appreciation to the Physicians, Surgeons, Hospital Executives and others who have so kindly consented to assist us in connection with the above program.

Note: The Censors will meet for the examination of candidates on the first Thursday of November, 1936 and May, 1937. Fee of $10.00 is payable at the time of examination. Application blanks may be obtained by writing the Secretary, furnishing name, address and name of school of graduation in medicine. Application must be made at least three weeks prior to date of examination. Candidates whose applications are on file will receive proper notices.

FRANK S. CRUICKSHANK, M.D., Secretary.
1247 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

October 15—11 a. m. at the Moore Hospital, Brockton.

FRED F. WEINER, M.D., Secretary.
231 Main Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

October 28 - April 28, 1937—See page 740.

November 5—Censors' Meeting. See page 740.

WORCESTER DISTRICT MEDICAL SOCIETY

November 5—At 4:30, in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the fall Censors' meeting.

November 11—Grafton State Hospital, North Grafton, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

December 9—St. Vincent Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

January 13, 1937—Worcester City Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

February 10, 1937—Worcester State Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

March 10, 1937—The Memorial Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

April 14, 1937—Worcester Hahnemann Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

May 6, 1937—At 4:30 in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the spring meeting of the Board of Censors.

Wednesday Afternoon and Evening, May 12, 1937—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

October 28—See page 741.

BOOKS RECEIVED FOR REVIEW

Diseases of the Nails. V. Pardo-Castello. 177 pp. Springfield and Baltimore: Charles C Thomas. $3.50.

Digestion and Health. Walter B. Cannon. 160 pp. New York: W. W. Norton & Company, Inc. $2.00.

Research in Dementia Precox. [*sic*] Nolan D. C. Lewis. 320 pp. New York: The National Committee for Mental Hygiene. $1.50.

The Riddle of Woman. A Study in the Social Psychology of Sex. Joseph Tenenbaum. 477 pp. New York: Lee Furman, Inc. $3.50.

The Clinical Use of Digitalis. Drew Luten. 226 pp. Springfield and Baltimore: Charles C Thomas. $3.50.

Roentgen Interpretation. A Manual for Students and Practitioners. George W. Holmes and Howard E. Ruggles. Fifth Edition, Thoroughly Revised. 356 pp. Philadelphia: Lea & Febiger. $5.00.

A Textbook of Obstetrics. Edward A. Schumann. 780 pp. Philadelphia and London: W. B. Saunders Company. $6.50.

Oral Diagnosis and Treatment Planning. A Text for the Dental Student; a Reference Book for the Practitioner and Medical Student. Kurt H. Thoma. 379 pp. Philadelphia and London: W. B. Saunders Company. $6.00.

A Text-Book of Pharmacology and Therapeutics of the Action of Drugs in Health and Disease. Arthur R. Cushny. Eleventh Edition, Thoroughly Revised by C. W. Edmunds and J. A. Gunn. 808 pp. Philadelphia: Lea & Febiger. $6.50.

Starling's Principles of Human Physiology. Edited and Revised by C. Lovatt Evans. The chapters on the central nervous system and sense organs revised by H. Hartridge. Seventh Edition. 1096 pp. Philadelphia: Lea & Febiger. $8.75.

Live Long and Be Happy. How to Prolong Your Life and Enjoy It. Lewellys F. Barker. 224 pp. New York and London: D. Appleton-Century Company. $2.00.

Anatomy of the Human Body. Henry Gray. Twenty-Third Edition Thoroughly Revised and Re-Edited by Warren H. Lewis. 1381 pp. Philadelphia: Lea & Febiger. $10.00.